INDUSTRIAL MEDICINE

The Journal of Occupational Diseases and Traumatic Surgery

MAY 21'41

May, 1941

The Pathological Process
— Following Fracture, and Treatment Methods —

Gall-Bladder Disease
— A Consideration of Mortality —

Traumatic Injuries
— and Mental Disturbances —

Cardiovascular Emergencies
— Recognition and Treatment —

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Trauma and Glomus Tumors
— Relationship, with Report of a Case —

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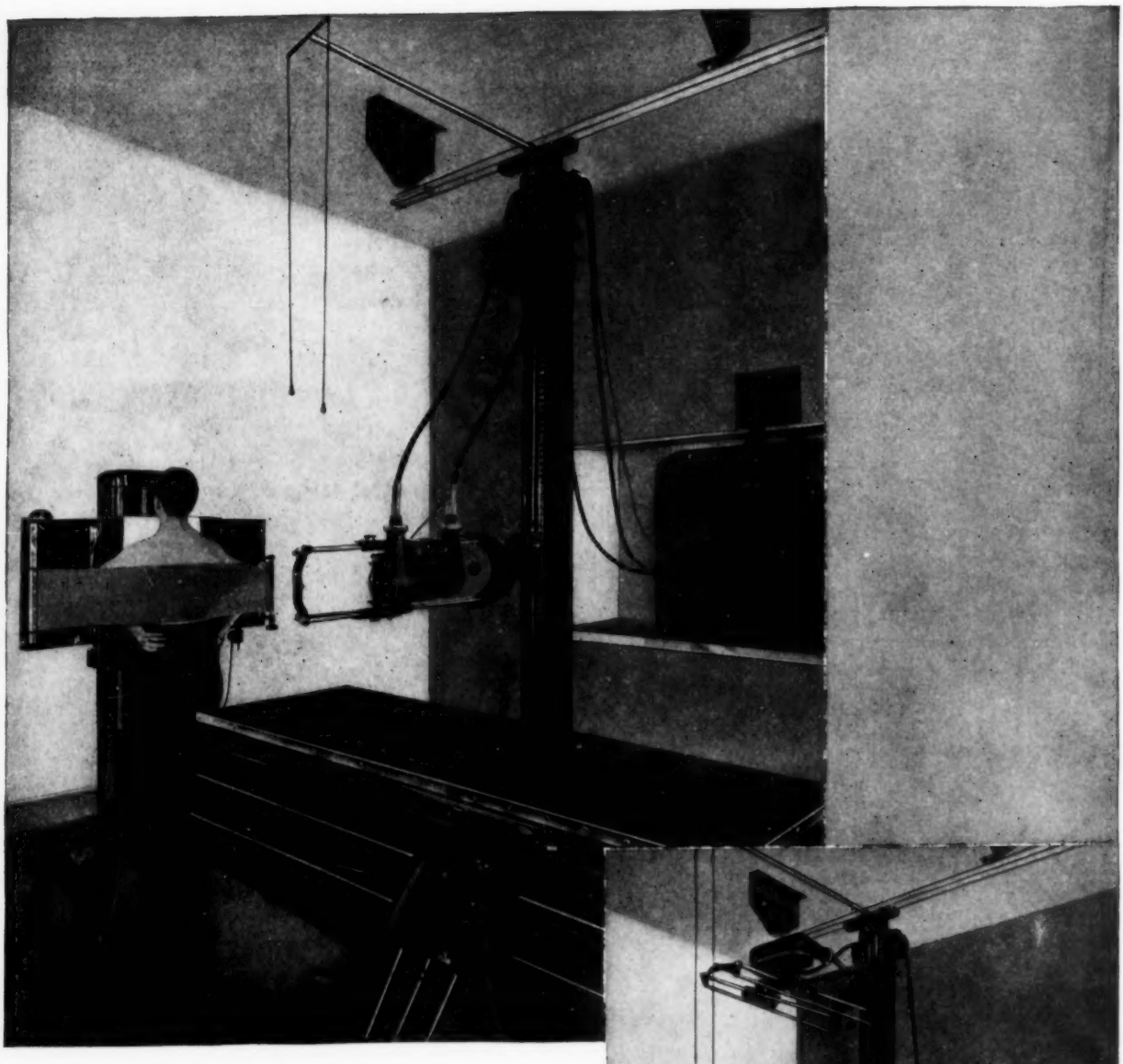

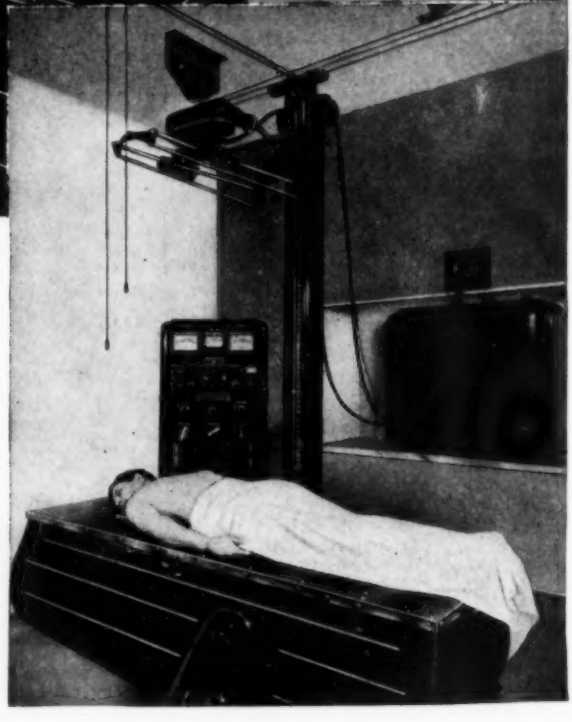

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THIS MONTH

THE PATHOLOGICAL PROCESS....CLAY R. MURRAY, M.D., F.A.C.S. 171
—Following Fracture, and Treatment Methods

ONE HUNDRED MILLION DOLLAR PREMIUM..CHARLES A. TOGUT 182
—Income for Workmen's Compensation in 1941

GALL-BLADDER DISEASE.....CHAS. GORDON HEYD, M.D., F.A.C.S. 184
—A Consideration of Mortality

TRAUMATIC INJURIES.....LEOPOLD VACCARO, M.D., F.A.C.S. 187
—and Mental Disturbances

EVALUATION OF MANIPULATIVE THERAPY..FREDERICK A. JOSTES 189
—in Back Conditions

CARDIOVASCULAR EMERGENCIES.....ROBIN C. OBETZ, M.D. 192
—Recognition and Treatment

THE INDUSTRIAL HYGIENE ENGINEER.....WARREN A. COOK 196

REGISTRATION of PHYSICIANS.....HUGH J. MURPHY 200
—Under the Workmen's Compensation Law, and Requirements for Advancement in Ratings and Qualifications

HISTORICAL MILESTONES....ALFRED J. WHITTAKER, M.D., F.A.C.S. 203
—in Occupational Medicine and Surgery
(with David J. Sobin, M.D.)

THE PITTSBURGH MEETING, May 5, 6, 7, 8, 9, 1941..... 205
—Program of the Twenty-Sixth Annual Meeting of the American Association of Industrial Physicians and Surgeons and the Second Annual Meeting of the American Industrial Hygiene Association

AMERICAN ASSOCIATION of Industrial Physicians and Surgeons.... 207
—Proposed Constitution and By-Laws Amendments

TRAUMA and GLOMUS TUMORS.....MARTIN J. LOEB, M.D. 208
—Relationship, with Report of a Case

WORK OUTPUT and PREGNANCY.....NORMAN J. POWELL 213

CENTRAL STATES SOCIETY..... 214
—Meeting, May 17, 1941

EDITORIAL: "In All Good Will"..... 216

INDUSTRIAL HEALTH and DISEASE:

INDUSTRIAL HEALTH and National Defense
LEVERETT D. BRISTOL, M.D., Dr.Ph..... 217
(In "J.A.M.A.")

DR. CLAY RAY MURRAY'S "The Pathological Process Following Fracture, and Treatment Methods" (page 171) is the transcript of a talk which was the feature of an evening meeting of the CENTRAL STATES SOCIETY OF INDUSTRIAL MEDICINE AND SURGERY. At our special request, DR. MURRAY permits us to print it as it was delivered—with the minimum of editing on his part, and the elimination only of certain reinforcements of emphasis here and there. In this way there is preserved for the readers, as nearly as may be, the sparkle and vitality of presentation which so vividly impressed the hearers. In substance, background, and authoritativeness the article is an unusual and momentous event; in form it indicates how very interestingly DR. MURRAY handles his subject, and accentuates the feeling, several times expressed on the floor of the meeting, and often expressed since, that talks by him are much too rare . . . MR. TOGUT'S "One Hundred Million Dollar Premium" (page 182) summarizes a situation which is of increasing importance not only to the physician but also to all others interested in workmen's compensation — employee, insurance carrier, employer, legislature, and commission. He makes one statement which some of the others now and then overlook: "Medicine is spoken through the mouths of doctors, none others" . . . DR. HEYD'S "Gall-Bladder Disease" (page 184) is having concurrent publication in the *New York State Journal of Medicine* . . . DR. VACCARO'S "Traumatic

—Continued on Page 6

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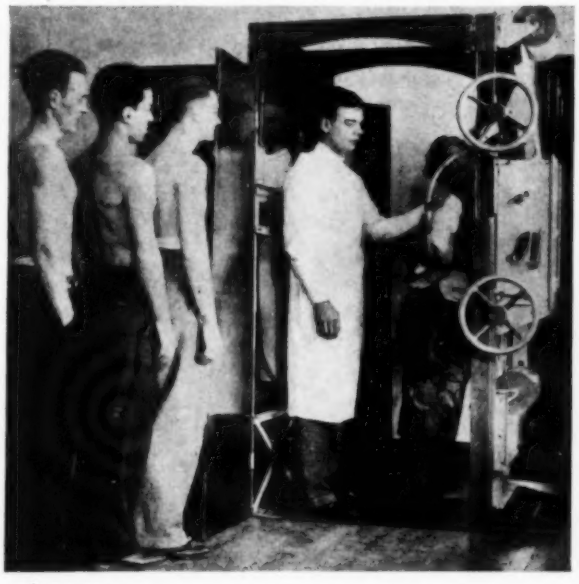

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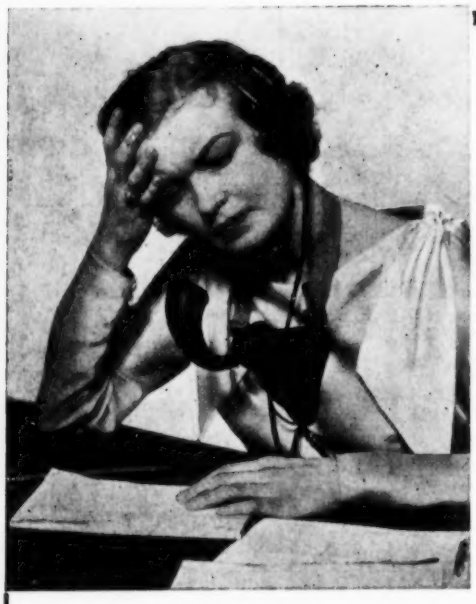

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—*Continued from Page 3.*

Injuries and Mental Disturbances" (page 187) is the second of two articles, the first of which was published in INDUSTRIAL MEDICINE for April, 1941. DR. VACCARO is Director of the Philadelphia Occupational Disease and Workmen's Compensation Medical Service. His percentages of disability, as summarized on page 187, have their basis in substantial experience . . . DR. JOSTES' "Evaluation of Manipulative Therapy in Back Conditions" (page 189) will be of inestimable value to many who have not had the opportunity for the careful study which this article comprises . . . DR. OBETZ'S "Cardiovascular Emergencies" (page 192) recognizes and discusses "one of the most important phases" not only of medical practice in general, but also of medical practice in industry. The subject is thoroughly and authoritatively treated . . . MR. COOK'S "The Industrial Hygiene Engineer" (page 196) was part of the Symposium on Availability of Trained Industrial Health Personnel, at the 1941 Congress on Indus-

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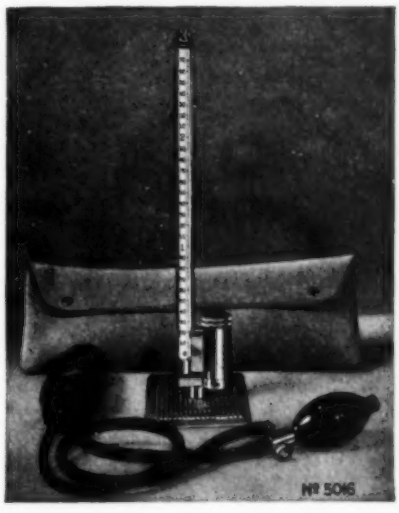

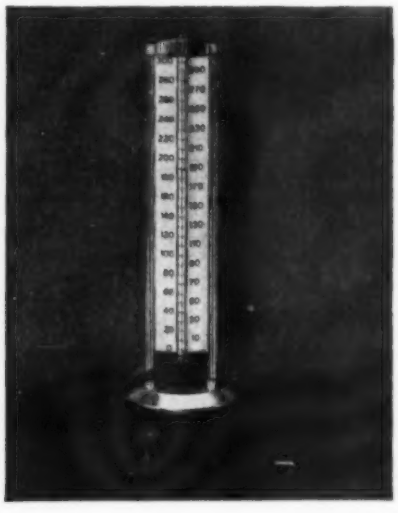

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trial Health. Hence the form of the treatment. His conclusions, on page 199, are particularly important. . . . MR. MURPHY'S "Registration of Physicians" (page 200) answers numerous inquiries as to the exact nature of the requirements in New York State, where "free choice" prevails. . . . DR. WHITTAKER'S "Historical Milestone" (page 203), concerning the work of Ulrich Ellenbog (1440-1499) discloses the interesting fact that only two copies of Ellenbog's book (1473), "the first printed thesis dealing with occupational health," are extant, and that he is the owner of one of them. We know of his interest in the early history of industrial medicine, and we hope that this exceedingly interesting "Milestone" is only the first of many from his library. . . .

The PROGRAM of the Pittsburgh Meetings follows on pages 205-6-7, together with the proposed amendments of the Constitution and By-Laws of the A. A. I. P. & S. which are to be voted upon at the business meeting. . . .

DR. LOEB'S "Trauma and Glomus Tumors" (page 208) reflects and exemplifies the quality of care and study that are necessary, in these days of many publications, to make an article a *real* contribution to medical literature. This is such a contribution, remarkable from many standpoints, and creditable in all. . . .

MR. POWELL'S "Work Output and Pregnancy" (page 213) is a begin-

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* Levin O. L., Behrman H. T., Industrial Medicine Vol. 10 No. 2 (Feb. 1941).

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ning study along lines that are certain to be of increasing interest to personnel administrators. . . .

THE programs on pages 214 and 215 are comprehensive in scope and vital in interest. Particularly valuable is that one announced by DR. LEROY U. GARDNER, of the Symposium on Tuberculosis in Industry, to be held at Saranac Lake in June. And no less, to those within its reach is that of the Western Association of Industrial Physicians and Surgeons to be held at Del Monte, California, on May 4.

THE Editorial (page 216) speaks for itself. It is followed by DR. LEVERETT D. BRISTOL'S painstaking analysis of "Industrial Health and National Defense" (page 217) which appeared in *J.A.M.A.* . . .

DR. W. W. LEAKE, of New Orleans, will become Chief Surgeon of the Illinois Central Railroad on May 1. His office will be at Chicago, and he will have charge of all medical service for Illinois Central employees, including the operation of Illinois Central hospitals at Chicago, New Orleans, and Paducah, Kentucky. He will succeed DR. G. G. DOWDALL who is

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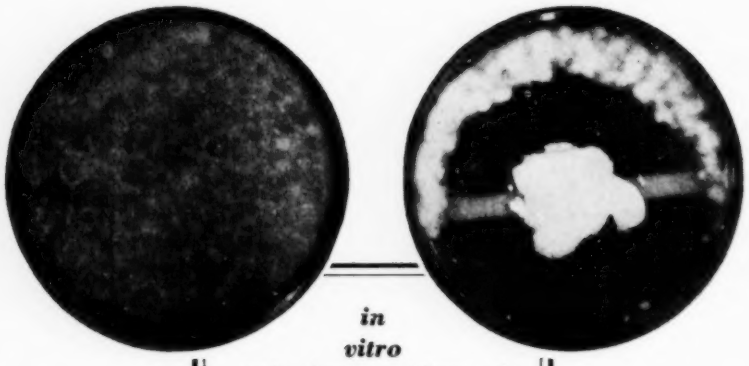

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nounces the third annual convention of group health plans, in June, for the discussion of possible approaches to the problem of "Health Services in a Defense Economy," with special onsideration of the matter of medical care for industrial employees. "Approximately 400 million man days are lost each year from absenteeism due to illness. About 20 of each 1000 employees are non-effective on account of illness; a greater number are only partially effective. The cost to industry approximates $60 per employee. More than 90% of absenteeism due to illness is caused by non-industrial illness."

As to physicians in industrial practice, *J.A.M.A.* says: "A listing of physicians shows by states the names of 465 physicians classified in industrial practice, 1534 physicians who specialize in industrial surgery, 48 who specialize in preventive medicine in industry, eight who indicate industrial consultation practice, and five who specialize in industrial toxicology. These lists are compiled from a total of about 95,000 punch cards and are not complete."

Air Hygiene Foundation's current letter in its series of "short reports for industrial health defense" is Report No. 3—"Fatigue in Industry" by Dr. J. R. Garner, of Atlanta, Georgia.

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NEW BOOKS

Bone Graft Surgery

Review by

FREDERICK W. SLOBE, M.D., F.A.C.S.

BONE GRAFT SURGERY IN DISEASE, INJURY, AND DEFORMITY, by FRED H. ALBEE, M.D., LL.D., Sc.D., F.A.C.S., F.I.C.S. Published by the D. Appleton-Century Company.

THE author, a pioneer in bone graft surgery, wrote his first book on the subject in 1915. This present work incorporates developments since that time, including both the author's own procedures and those of other surgeons which, in Dr. Albee's opinion, are the most meritorious.

The first chapter is devoted to the general principles of bone grafting, dealing with vascularization and life of the graft, and the relationship of coaptation and immobilization to union. The graft not only lives but increases in size according to Wolff's law. The vital importance of biological and physiological principles in bone grafting for non-union in addition to mechanical principles is emphasized. In this connection, the author feels that the intramedullary, osteoperiosteal, and onlay grafts are inferior to his method.

The armamentarium is discussed in a practical chapter of 40 pages including instruments, materials, fracture table uses, etc. A very ingenious series of diagrams exhibits the mechanical analogies of standard bone graft procedures.

The third chapter is devoted to spinal fusion and the various conditions for which it is indicated. The tibial bone graft is preferred, the reasons being the poor osteo-

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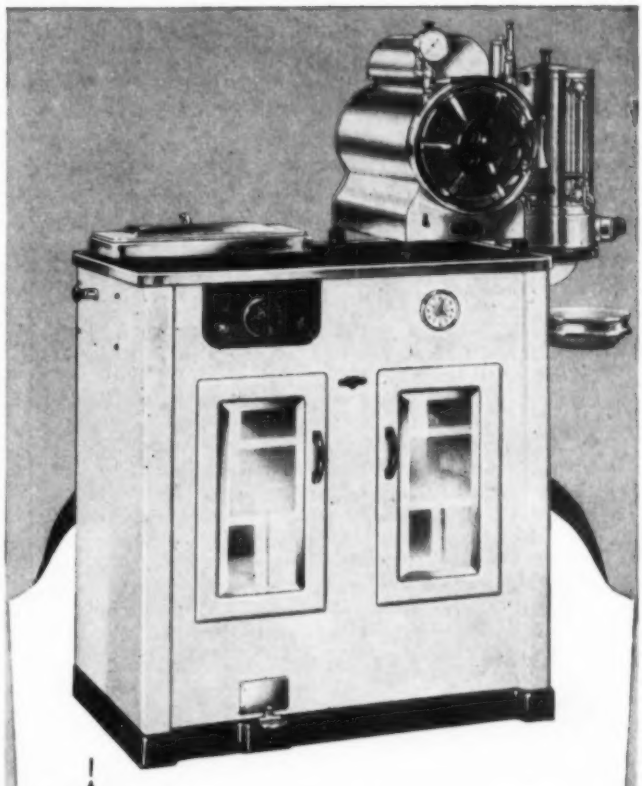

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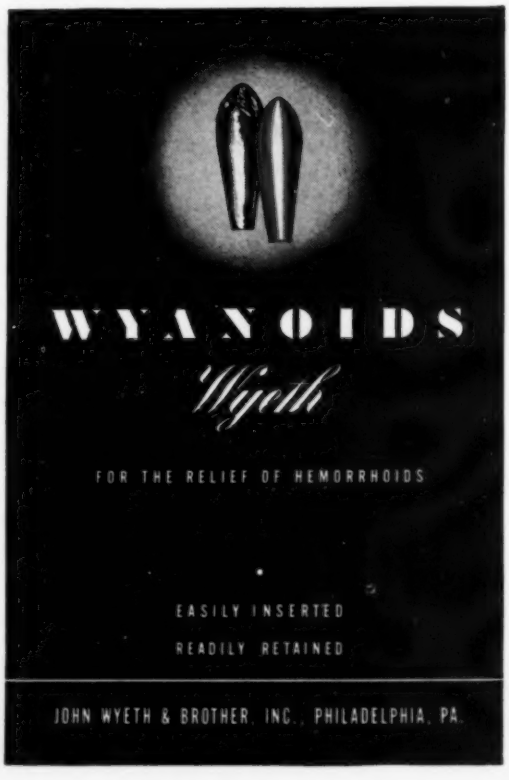
genetic quality of vertebral bone and the very favorable effects produced by immobilization and revascularization. The inlay graft technic is amply described both by text and illustrations. Other types of fusion procedures devised by various surgeons for special conditions are described including those of Girdlestone, Hibbs, Mercer, Speed, Mathieu-Demirleau, Campbell, and Gaenslen.

The next chapter (devoted to the hip-joint) is of particular value because of the current interest in the operative fixation of fractured hips. Adhering to the opinion that loss of blood supply is the chief cause for non-union in intra-capsular fractures, the argument is advanced that the bone graft peg is biophysiologically correct by furnishing osteogenetic callus-forming material and acting as a "vascular conducting scaffold to the anemic proximal fragment" as well as immobilization. In his reconstruction operation the mechanical effectiveness of the bone-muscle lever produced by his procedure is likened to that existing in various animals, illustrated by diagrams of comparative anatomy. Shelf operations for congenital dislocation and various procedures for extra-articular arthrodesis are shown with complete diagrams and illustrations. The discussion of the principles of the abduction lever are especially interesting with operative correction well described and the indication therefor.

Obviously, the section on ununited fractures is one of the important features of this work. Inlay grafts, sliding inlay grafts, double-wedge-end grafts, sliver grafts, and bolt grafts are described. The technic of the onlay graft of Henderson and Campbell is given. Regional modifications for special bones as the clavicle, patella, olecranon, scaphoid, calcaneous, and jaw are shown.

A special chapter is devoted to more extensive procedures for replacement of bone by grafts. Bone tumors, cysts, osteomyelitis, and trauma may furnish such indications as does congenital absence. It is here that some of the most striking results are obtainable in rehabilitating hopelessly functionless members.

Plastic surgery is covered in 50 pages to include the replacement of cranial defects, correction of nasal deformities, jaw reconstruction, synthetic formation of digits, leg lengthening, arrest of leg growth, and even enlargement of the parturient canal. Bunnell's method of reconstructing the thumb is given in detail.



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The last chapter, in describing bone block operations, quotes from numerous surgeons including Campbell, Mayer, Eden, Speed, Putti, L'Espiscopo, Miltner, Dilger, and others in discussing procedures blocking the knee, angle, shoulder, jaw, and hip.

Throughout this book, a definite aversion to the use of metallic fixation and non-absorbable sutures is manifest.

The work covers 403 pages and is replete with illustrations, diagrams, and roentgenographic reproductions. After each chapter a bibliography is appended. Each page of this book is solid, concise discussion and, as a consequence, a tremendous amount of information is contained therein. Written by one who is a pioneer and master in this field, the originator of numerous operative procedures, and the designer of many special instruments, and representing many years of surgical research—it is a most noteworthy contribution to surgical literature.

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Industrial Medicine

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The Journal of Occupational Diseases and Traumatic Surgery

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The Pathological Process

—following Fracture, and Treatment Methods—

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SINCE the study of the pathology of the fracture lesion has been seriously made, it has exerted very far-reaching effects on modern fracture treatment. The basic principles of all fracture treatment are absolutely dependent upon knowledge of the pathological process throughout its course.

I might start in, perhaps, by saying something about compound fractures and utilizing purely the knowledge we have gained by the study of the pathological process and its timing sequence in this lesion and its effect upon treatment methods.

Time was when it was presumed that at the mention of iodine, every organism within hearing dropped dead. That time has passed. We know today that antiseptics strong enough to kill organisms will hurt tissue. Antiseptics are being gradually discarded, if we may except the present questionable enthusiasm for sulphanilamide and sulfathiazole, in the treatment of compound fractures unless, by reason of misadventure, they have passed the point where they can be given adequate treatment.

In dealing with a compound fracture, we are dealing with two things: first, a fracture; second, the question of infection. The fracture should be treated according to the rules that hold for all fractures, whether they are compound or not compound. The compounding affects only one element in the story, and that is the question of infection.

At the time of contamination—if we are to discriminate between the terms "infection" and "contamination"—the organisms are on the surface of the tissues; not on the surface of the body, necessarily, but on the surface or surfaces of the wound. That is true whether the fracture is compounded from within or compounded from without.

I have always found it extremely difficult, on inspection of a compound wound which happened a few minutes before, and in which the bone has punctured the skin and then has withdrawn again into the depths, by my clinical judgment to decide whether hemolytic streptococci of a virulent form have gone into the wound.

There are some men who can do this. They can look at a compound fracture, compounded from within, see a centimeter-long skin slit, fail to visualize the four-inch hiatus into the tissues below that centimeter skin slit, and be absolutely certain and secure that since the bone made the slit in the skin and then hurriedly withdrew, it was polite enough to leave the hemolytic streptococci on the skin and to not bring them in. So, they rule that such wounds should not be debrided because they are compounded from within and, therefore, are perfectly harmless.

That is the wish being father to the thought. Those fathers bear frequent monstrosities. There is no such thing as a distinction between a fracture compounded from within and one compounded from without. A compound fracture is a compound fracture, unless it has been made in a blast furnace where there aren't any organisms. The risk is just as great, or may be in that particular case just as great, with a half-centimeter skin puncture as it is with a four-inch wound—sometimes greater.

The answer to that, of course, is obvious: "He is talking through his hat. I had a case just last week. I sewed him up, didn't even bother to put a drain in, and look at the man. He is fine."

Gentlemen, it never pays to boast about what you got away with. It leads you into a fool's paradise. I will illustrate that with some actual cold, hard, real, not Rooseveltian statistics on compound fractures in just a moment.

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So, on the basis of this pathological study, we can say that there is no distinction between a punctured wound with a compound fracture and a wide open compound. They both should be treated by what? By debridement. That is the answer. Any student knows that.

What do you do to a compound fracture? I debride it. What do you do when you debride it? Well, I eliminate all dead spaces, where hematoma or pocketing could occur. I remove all dead tissue and save all living tissue. I eliminate all tissue that seems to me devitalized and whose death is probable. I secure as complete hemostasis as I can.

Should you always debride a compound fracture? Raised eyebrows and, "Why, of course," as an answer. That is all wrong.

When do you debride a compound fracture? That is answered in minutes, not in hours. The debridement of a compound fracture is based on the assumption that you can remove from that wound the organisms, the dead spaces in which they can live and multiply, and the tissue on which they live, which is dead tissue or dying tissue, devitalized tissue.

You can take the dead tissue and the dying tissue out if you wait a week, and you can eliminate the dead spaces a week later. But, you can not get rid of the organisms unless they are still on the surface. All you can do is open up new channels for the organisms which have penetrated the tissues to extend still further.

So, for a debridement to do what it is supposed to do, it has to be done before penetration of the tissues by the organisms occurs. That is why I say that it is a problem to be determined in minutes after injury, and not hours after injury. So much so, that a good many of us are now quite convinced that after a period of 12 hours following a compound fracture, it is highly questionable as to whether any debridement ought to be even attempted in a wound.

If drainage is needed at this time it can be established. You have an infected wound. There is no question as to whether or not you have a *contaminated* wound. You have an *infected* wound, and you can not get rid of the infection. The patient, if you give him a chance, can; but, you can't.

If you see the patient while his organisms are still on the surface, before they have penetrated into the sound tissues, you can get rid of the organisms. After that time, the patient is the only one who can get rid of them. And, it is very questionable as to whether you help him any by traumatizing his tissues still further at that date and opening up areas which may not yet have been invaded.

Therefore, late debridement is, to say the least, a questionable procedure. A late compound wound should call for adequate drainage, elimination of dead spaces, but without actual dissection into the tissues.

Two things have developed from that study. One is the elimination of the late debridement as a harmful rather than a beneficial procedure. The second is estimating the time in minutes from the accident to the operating room instead of hours. If I were asked to pick the one factor which exerted the greatest influence in deciding whether or not a compound wound would become infected, I should pick as that one factor the number of minutes between the accident and the time the patient's wound was adequately treated. It is a factor which frequently is under our control.

Now, this may sound a bit astonishing to you, but in the Presbyterian Hospital today, if an acute appendix and a compound fracture were to come into the hospital coincidentally, and only one operating room was available, the compound fracture would go to the operating room and the acute appendix would wait until the fracture was done. That is not theory; that is fact.

It is logical and sensible, because in two hours the purpose for which you are sending the compound fracture to the operating room may be completely ruined. You haven't a chance. Your hands are tied before you start. You can't accomplish anything. Two hours in an acute appendix, I believe, never made a tremendous difference in a patient's welfare. We have taken it that seriously because we believe it is that serious a problem.

NOW, when it comes to the question of what to do with a wound, the problem is: first, do as little damage as you can on the principle that if you can not help, don't hinder. In other words, your debridement should be as gentle and as free from trauma as possible. Use nothing but a sharp scalpel and a pair of forceps. Do not use scissors, which crush tissue, but use a sharp scalpel and a pair of forceps, plugging the wound first, shaving and cleaning the skin thoroughly.

Then, if you have a particular esthetic value which demands that that skin be pink or brown or blue or yellow, indulge in it—but, make the medium that gets the color weak enough so it can not hurt the tissues. It may stagger a few bugs, but don't depend on it. By the next morning, they will be feeling pretty good. The big point is: don't hurt the tissues because you are going to have to leave bugs there.

You can not, even in an operating room, create a sterile wound. Some bugs are going to be in there, and, no matter how much you may think of yourself, it is the patient who is going to have to deal with them. He is going to have to deal with them with the tissues on which you are going to spill these beautifully colored antiseptics. Don't harm them. Leave the tissues their full strength to deal with the end of the problem that you can't deal with.

You can do something to eliminate organisms if the debridement is done when they can be eliminated; that is, you can wash them out. Just as when you spill coffee on your vest, you can rub it off right away. But, if you discover it an hour later, it has to go to the dry cleaner's.

The best thing for washing contamination out of tissues is the thing that mechanically works, and which irritates the issues least, which is a normal physiological solution, saline. And you don't use

teaspoons full of it. You don't take a little pot and pour it into the wound. You start your measurement in gallons; and the only limit that I know of is your patience and the amount of saline. You can not use too much. The more you wash, the better chance you have of insuring a wound which is not going to succumb to infection.

You wash from the bottom out. You don't wash from the outside in to see how much you can wash down into the tissues. You put a soft rubber catheter down in the bottom of the wound, and the flow is from the inside outward. I mean, literally, gallons of solution.

Now, when you have done that, one thing you can say is: "I have done a minimum amount of harm to this patient." Believe me, that is a wonderful confession to be able to make.

Second, you can say, "I have eliminated all the dead tissue which I can find, all that I think is dead, and all the tissue which I think is going to die. I have left no dead spaces, and I have spared all tissue which is viable, so far as I can tell."

Well, there ought to be some fun in this. So, let's make a little bet with the patient. Of course, he is unconscious, but he wouldn't object to the bet if he were conscious. I am going to bet my surgical judgment against his leg. The problem is whether we will close this wound or not. Of course, I might be wrong, but we will make it a sporting thing. So, I will bet my judgment against his leg, and I am going to close this wound. It is not a swell bet for the patient because it is heads you win, and tails, he loses. He hasn't got a chance in the world. If he gets well, you are a wonderful doctor. If he doesn't, it is just a tough break.

Elsewhere in the body, if we were to introduce bugs into the tissues and wanted to be sure they would grow, we would create tension in the tissues. It would be an ideal set-up for creating infection. We know that when any portion of the body is injured by trauma, it swells; hemorrhage sets in; edema; inflammatory exudate; and the tissues swell. Swelling causes tension, provided it is confined.

So, realizing that we are dealing with a wound in which we may still have left some dead tissue and in which certainly there is traumatized tissue, and in which we know we have left some bugs, with the wound wide open there can not be any tension because edema and exudate just leak right out into the wound.

We don't consider that a sporting proposition. That doesn't give the bugs a chance. So, we close the wound so that when the swelling occurs it will be confined, and there will be tension in the tissues.

Now, that reductio absurdum is something, of course, to laugh at. You know you have seen it time and time again. Again, boasting about what you get away with. "Patient sewed up; no trouble at all."

They ran 200 cases at the Harlem Hospital when Jack Connor was alive a few years ago; two series of 100 cases each. Each was a case of compound fracture which, in the opinion of the surgeon, it was safe to sew up.

In one series of 100 consecutive cases, they placed sutures, left the wound open, and, as soon as the edema induration and infiltration of tissues had disappeared, they did a secondary closure on wounds. In the other series they closed all wounds.

In the cases in which the surgical judgment of the individual had decided that the wound should be closed, and it was closed, they developed six infections in the hundred.

In the cases which were left open, they developed eight infections—two more. Now, that comparison shows I am talking through my hat right now because I don't believe you ever ought to close any compound wound except under very exceptional circumstances, and yet the open wounds showed the most infections.

But it comes to analyzing the cases that you didn't get away with. Of the six cases in the hundred that were closed and developed infection, one died of general sepsis; one lost a leg through amputation from sepsis; and all but one had chronic osteomyelitis.

Of the eight cases who developed infection in the open wound, without tension and complete free drainage from the beginning, no case developed sepsis; no case had amputation; only one case developed osteomyelitis.

So, it isn't a question of what you get away with. It is a question of what happens to the poor, unfortunate individual you don't get away with. To my mind there aren't any odds to this story. There is no question about it.

If any man can show me that in a compound fracture you will lengthen the patient's convalescense or cause him the slightest risk of his fracture result by leaving a compound wound packed open, never inspected from the time it is operated on; not exposed to frequent dressings, and secondarily closed as soon as the inflammatory reaction has disappeared—the minute any man can show me that a patient's convalescence time is lengthened or his fracture healing is interfered with by that procedure, I will change my mind and close compound wounds.

But, I will be damned if I will do it before! I have never seen a case that had his convalescence prolonged, his healing interfered with, by leaving the wound open those few days. Believe me, I sleep a lot better nights when they are open.

I THINK that lesson is becoming more and more true. The reason I mention it tonight specifically is because of the increasing use of sulphanilamide, sulfathiazole, and various drugs of that series in compound wounds. I saw such a series of cases recently, only a dozen of them. They all got well. It wouldn't be the first time I saw 12 compound fractures in a row sewed up and get well. I have seen them without sulphanilamide get well. What I want to see is a big enough series of cases to find out what happens to the ones that don't get well that are sewed up. I have a terrible hunch that it is going to be the same story as when they are closed with surgical judgment instead of with sulphanilamide. I don't believe that the mere intro-

duction into the tissues of that drug is a sufficient safeguard with closure.

Rather interestingly, the first figures that I have seen—I may be behind the times on this—but the first figures I have seen on the actual concentration of sulfathiazole in a wound so treated, I got from recent series which is being carried out at the Harlem Hospital in New York, one patient with a compound wound being given sulfathiazole by mouth; and another patient who had had it put in the wound.

There was some fluid accumulation in the one who had it put in the wound. Incidentally, there is definite evidence tending to show that the presence of these two drugs interferes with wound healing and produces a considerable amount of reaction and edema in the tissues. But, the concentration of sulfathiazole in the wound in which it was placed, as determined by experimentally drawing out a syringe full of fluid, was 648 milligrams per cent. In the patient whose bloodstream findings were 3.8 milligrams per cent, after mouth dosage, his wound findings were 3.8 milligrams per cent.

So, the mouth administration, apparently, gave a 3.8 milligrams per cent concentration in the fluid in his wound with very little in the way of local reaction, in the way of edema, and so on, beyond the normal. But, the sulfathiazole in the compound wounds, put directly into them, and with the wound closed, showed 648 milligrams per cent in the wound. This was four days after the thiazole was put into the wound. So, it apparently does reach a tremendous concentration and dissolves to that extent into the tissues. That patient's blood level, by the way, never went above 4.2 milligrams per cent despite the tremendous concentration in the wound.

However, I think a word of warning about accepting too readily the complete safety of putting some sulfathiazole into a wound, sewing it up tight, and forgetting about it, is not untimely. I think it is something to be considered purely experimental at the present time. I don't believe there is nearly enough evidence at the present time to warrant its general use in uncontrolled cases where careful study of both the patient and the wound is not being made.

I COME now to another factor in which pathology has been studied. There has been a perfectly tremendous tempest in a teapot for the last five or six years about what is known as the electrolytic reactions of metals in tissues. You would think with a piece of metal in them, to hear some of the conversations and discussions that go on, that a patient who had an internal fixation of a fracture walked around a room shooting vivid sparks a mile long every step he took. It is nonsense!

Ninety-five per cent of the difficulty that metals of the steel group cause in the tissues have nothing to do with the metal whatsoever. They have to do with the fact that the metal is either: one, not rigidly fixed in the tissues; or, two, that the fixation allows it to be subjected to the stresses and strains which cause physiological repercussions in the tissues in the form of inflammatory phenomena, marked increase of acid pH of the tissue fluids, and, therefore, absorption of any bone that happens to be bathed by those tissue fluids.

We had a patient at the hospital who had a fracture of both bones of the forearm. We operated on him. One bone was rigidly fixed, long plate, screws that went through the opposite cortex and a cross transfixion screw. By that I mean, in addition to that one plane fixation, which has very little control over torsional strains and practically none over strains against the plate instead of with it, two transfixion screws were put obliquely through at right angles to these, from one fragment out the opposite side of the other. That took care of the torsional strains and strains against the plate.

The other fracture—you know the old adage: "In fracture of both bones of the forearm, forget the ulna and look after the radius." It is fallacious, pure and simple. Anybody who follows the cases will find that out. We worked on that basis. We used a short plate with screws that didn't go through the cortex, no transfixion.

We operate on a peculiar basis. We don't operate for position alone. I may as well stick my chin out: we operate on a case of a fracture, for instance, of both bones of the leg in an adult that comes in in perfect position, in order to get it rigidly fixed. But that is because we are not operating for position. We are operating for convalescence time. By the time the patient who has been plated is healed, he will have complete and full knee, ankle, and hip motion. When his bone is healed, he is healed; instead of by more conservative methods, starting to heal the patient after you have damaged him by healing the bone.

We can cut convalescence time in two by doing this. But in order to do it you have to have rigid fixation. You can't use fixation that develops stresses and strains against the plates and screws that are used, or whatever fixation materials you use.

I am sure Dr. Jackson agrees with me. In many cases he operated on, he could have got position by other methods. Dr. Jackson operates for convalescence time and rapidity of convalescence. He stresses it in his patients. Yet when he gets a long spiral fracture, he puts bands around it, which necessitates his tying it up in a plaster until the bone is healed, although he has perfectly good plates and screws right at hand to do it with that would make it unnecessary to tie the leg up in plaster.

The principle of the thing is early active motion, which is possible only if you get rigid fixation—he brought that point out splendidly. Sometimes a man can say, "Well, we saw some patients today who did not have that cross fixation. They got away with it." Again, that is boasting about what you got away with; and, as I say, it is a dangerous thing to do. Take enough of these cases, follow them long enough, and you find unless you get that rigid fixation, it is not safe to start them using the

part. Screws will pull out; absorption will occur; plates will break; this, that, and the other thing will happen. Therefore, rigid fixation is essential in order to get early, active motion.

Absorption occurs because the screw is not in a table leg. It isn't even in a dead calf bone. It is in a living human being, who has physiological responses. If you subject bone to an inflammatory reaction, you will get decalcification of the bone. If you take a piece of metal and sink it into the bone and subject it to stress and strain unopposed, you are going to get absorption of the bone in the region of that stress and strain. You can't get away from it.

The only way it can be avoided is by having the fixation such that those stresses and strains are distributed so that they don't have to be met by any one single point of fixation, one single plane, let us say, of fixation. That has a pathological basis. We have sections of these screw holes under various circumstances.

The patient I talked of with fracture of both bones of the forearm—mind you, same patient, same operator, same arm, two bones. The radius which was rigidly fixed has a plate that isn't going to be touched forever, so far as I know. We have had plates in 28 years. There isn't evidence of the slightest bit of absorption in any of the screws and plates. Right alongside of it is the little apology for fixation in the ulna, with absorption around every screw hole and around the whole plate. The same patient, same metal, same electrolytic reaction — but it is fixed in one place and it isn't fixed in another. And, that is the answer.

Now, with a very small amount of electrolytic reaction from metal in a tube of Ringer's solution and with the production of an amount of oxide that you could put in a flea's eye and he would scarcely blink, one can color the Ringer's solution a dirty brown. It doesn't take much. It doesn't mean anything to look at the brown solution. It doesn't mean a thing, except that it is very theatrical and very impressive. It represents micrometric measurement.

On the other hand, it isn't a question with these metals as to whether or not they cause electrolytic activity in the tissues. The question is: Do they cause *enough* electrolytic activity, per se, to cause any disturbance in the physiology of the parts or of the healing process?

The answer for practically any metal in the steel group is no, if they are rigidly fixed. Vitalium is supposed to be the metal par excellence. It is actually stable in the tissues. It has no action whatever. Of course, you can't move a leg or an arm in which you put a vitalium plate because vitalium is a cast metal. I have x-rays of vitalium plates with at least 24 casting effects in one plate. If they move, they may break the plate, so you may have to tie them up in plaster. That is one reason why vitalium can not be used with mobilization.

Secondly, one can take the most stable vitalium plate ever used, put it in a bone so it is bearing stress and strain without rigid fixation, and I will guarantee you absorption about every screw you put in. That is how important I think the metal is. I shouldn't say think—we have evidence of case after case, clinical evidence, experimental evidence, pathological evidence, to show that rigid fixation is the factor that is important, if you use a metal in a steel series.

If you go into brass and copper, pure nickel and things of that sort, then you have real chemical irritants in the tissues. But in the steel series there isn't any one metal that I know of that causes enough electrolytic reaction, per se, in the tissues to cause any interference with bone healing.

I think a great deal of stress has been laid on the electrolytic action on the basis of test-tube colorimetric determinations. That is not tissue pathology. That is vivid imagination. If you have a string galvanometer with an amplification system such that the galvanometer swings in an arc half across the room, the effect is tremendous.

It doesn't mean a thing! I think that we can dismiss irritation caused by the metal, per se, in the tissues as, to say the least, a very much overrated factor.

What it means is that the man whose plates and screws cause trouble blames it on the metal instead of on his own ineptness in putting them in. So long as he continues to do that, he will continue to put them in poorly. When we understand that a plate, rigidly fixed, will not cause trouble, and, if trouble occurs, it is because the mechanical conditions make it impossible to fix rigidly—we will recognize perhaps an error in operating judgment, or an error in mechanical deftness. But it can not be blamed on the metal. That alibi has been demolished pretty completely.

NOW, I would like to touch upon another factor, the question of delayed and nonunion. It is perfectly fascinating, the schedule that this fracture healing process has. I don't want to enter into any discussion of how bone is formed. I wish I could even say I knew. I don't believe there is such a cell as an osteoblast in the adult human, if by osteoblast you mean a cell intermittently and specifically concerned in the formation of bone.

I think bone formation in the human adult is a biochemical accident, which is determined by the presence of undifferentiated connective tissue in the adult human body, where it only appears ordinarily as a result of injury as a replacement tissue, and by the presence of a pH in the tissue fluids which approximates the normal or even runs to the alkaline side, the high side, and by the local presence of calcium.

In fact, one can experimentally, by producing those conditions, produce bone at any distance from bone without ever having had contact with it. It is not difficult. It can be done in muscle. It can be done in fascial planes. All you need is calcium, undifferentiated connective tissue, and the proper pH. All you have to do to prevent the bone forming is to keep the pH acid. Let the pH, with the other factors present, go up to normal,

7.2 or 7.3, or beyond that on the alkaline side, and calcium will deposit in the new undifferentiated connective tissue to form bone.

Let that tissue differentiate into formed connective tissue, and you can't form bone in it.

You can have all the calcium you want there, and unless it causes sufficient inflammatory reaction to kill the tissue, and thereby give rise to new undifferentiated connective tissue to replace the dead tissue, no bone will be formed. That is possibly one reason why you see calcification in adult connective tissue as a degenerative process, but you never see bone formation. You only see bone formation when there has occurred undifferentiated connective tissue production. What is more, to get that bone formation, there have to be present at the same moment three things: Undifferentiated connective tissue; calcium, of a type which can be utilized; and proper pH conditions in the tissue fluids.

This is all hypothetical now, and has to be pinned down to facts in just a moment. Let's see where it leads us. Let's consider ultimate interference with the circulation—when I talk of circulation, I mean lymphatics, capillaries, and tissue spaces that hold the tissue fluids. That is the essential circulation of fracture healing because the pH of tissue fluids is the determining factor.

Lets' interfere with that circulation so there is some stagnation and the products of tissue death and the products of tissue metabolism are not carried away. The net result is what? A persistence, permanence of acid pH in the tissue fluids.

We have perfected a mechanism whereby we can, within an accuracy of two-tenths of a pH degree, test the pH of tissue fluids in vivo by using a very fine Corning glass, and a radio amplification system for recording differences in potential, translating those against known buffer solutions into variations in pH. We have been able to determine that at the time of fracture, as a matter of fact within two hours after fracture, the pH of the tissue fluids at the fracture site, may be as low as 4.2.

Within 12 days that pH, under ordinary circumstances, has returned to a pH of normal or, sometimes, as high as 8.2.

The same electrodes, placed in normal thighs at a distance from the fracture, will give no difference in potential whatsoever. We can check against the two sides of the body and against buffer solutions, so there isn't any inaccuracy.

We have been able to show by x-ray that while acid pH is present a decalcification of the bone in the region of the fracture occurs. We have been able to show that within six days of fracture, there is over 250 times the normal calcium concentration in the tissues immediately surrounding the bone. We have been able to show that this pH calcium concentration doesn't extend more than a few millimeters from the bone; and a half a centimeter away from where the pH in the tissue fluids is 4.2, the pH of the tissue fluids is perfectly normal. It is a local phenomenon associated with acid pH locally in the tissue fluids. It has nothing to do with the major circulation except very indirectly.

Now, if that pH, because the circulation which carries away the acid products of tissue death and tissue metabolism is interfered with, stays acid, or if tissue becomes formed, differentiated connective tissue, you can't get bone formation, and you have non-union.

It is a biochemical phenomenon, and the treatment should be directed to what? To improving the local circulation tissue fluid and capillary circulation. Empirically, everybody has been doing that progressively for the last 20 years by devising methods that allow of early active use of extremities to develop joint motion, to get back function.

But, they all add that union seems to be better when they do that. Of course it does! What they are affecting is the very mechanism by which capillary and lymphatic and tissue fluid circulation is kept going, and that is muscular activity. Blood pressure doesn't keep it going. Pure hydrostatic laws don't keep it going. Muscle activity is the major factor in maintaining adequate minute circulation, and all the efforts which have been expended in improving fracture treatment so that active function can be used from the standpoint of rehabilitation of the patient have coincidentally markedly improved the character of the healing in the fractures in which those methods are used.

Therefore, on that basis, can be explained an undisputed fact which has heretofore been rather puzzling. I think I can state this as a flat fact:

Any fracture which is subjected to open reduction and is then immobilized in plaster takes longer to heal than a similar fracture treated by closed methods. Any fracture which has been operated on and which after operation can be mobilized within the first 72 hours will present a shorter healing time for the bone than a similar case treated by closed methods.

It isn't a question of whether you operate on the case or not which decides union. It is whether you move the leg or the arm after operation that gives you union; and whether you can move the leg or the arm depends on whether or not it is rigidly fixed at operation.

There are a lot of other explanations given for that story, but it is almost inescapable on the evidence presented country-wide, not only country-wide but world-wide, on the study of these cases, that those statements I have just made are true.

WE HAVE not had in five years, since using this method of rigid fixation, one single case of non-union in operated clean cases of both bones of the leg. We operate routinely on every fracture of the long bone in an adult that comes into the hospital from the standpoint of convalescence time entirely.

What is more, we have cut our x-ray evidence of union time in tibias from an average of seven months—and by x-ray evidence, I mean absolute obliteration of all fracture lines—to an average

of four months. These are cases which are followed for at least two years. That is the minimum follow-up time, and no case that has not been followed that long is counted in that evaluation.

Nobody can delude me into believing that this story is a question of the man who does the work except for one thing: he has to know how to put a screw into something with a screwdriver; and he has to know the size of the drill hole that a screw calls for; and he has to know that when he drills the hole, if the drill wobbles a straight screw is not going to fit the resulting conical hole and be tight. When he uses a screw-driver, he has to know that when he lets the screw wiggle in the screw hole as he is cutting the thread, it is not exactly a precise mechanical job.

Believe me, to get rigid fixation, you have to be precise mechanically, and damned precise. If it is done that way, I certainly would say on the basis of the study of pathology involved, that operative methods today offer, where they can be carried out, the ideal method of treatment for fractured long bones in adults.

Dr. Sherman, you know, believes, as Dr. Jackson, Dr. Dyer, and I, that more of it should be done. He says, "There is only one way to treat a fractured shaft of the femur. Operate on them, plate them, start them on active motion. If you don't believe it, I will show you some."

He trots out 300 cases that have been back to work in the steel mills in four months. John Smith, who is attending this clinic, comes from an industrial town of, say, 25,000 people. He has been using rest and traction on all these femurs, and he thinks "There is something there. You can't laugh those off." So, back home he goes, and operates on the next 25 femurs. Well, he gets a couple of nasty infections and a couple of non-unions. Most of them take longer to heal than if he treated them by rest and traction, so he sits down and writes a paper for his county society. "If you have a patient you don't like, and you want to play a nasty trick on him, and he has a fractured femur, operate on him."

They are both right. Only, what Sherman means is this:

If you have my skill, my experience, my mechanical knowledge; if you have my assistants, who have been with me for 10 years; if you have my operating room assistant, who has been with me 10 years and knows every tiny detail of my operative technique; if you have my armamentarium, which consists of everything money can buy to make this procedure easy, non-traumatic, and riskless; and if you have my patient, who is a 'Hunky' in the steel mill that you can beat on the head with a mallet and he brushes it off and goes about his business—operate on him. He is absolutely right.

What Dr. Smith means is: If you have my knowledge and skill, which is that of an average surgeon with no special training; if you have my assistant, who is a house surgeon on a rotating service, three months in the operating room and this is his first week; and if you have my nurse, who rotates through my operating room for six weeks, and this is her second week; and my equipment, which is that of the average small, general hospital; and my patient, who is a bookkeeper, who weighs a hundred pounds in his winter overcoat and the only exercise he ever has is to take the dog out at night—for heaven's sake, don't operate on him. And he is right.

Those safeguards, those provisos, when we talk of operating, must be made. Those are important criteria. In my own opinion, it is 25% man; and it is 75% organization and equipment. You can't stand off and say, "I am just as good a surgeon as that fellow over there. If he can do it, I can." That is only 25% of the question. The real question is: What do you have to work with? That is the answer usually, and the deciding answer, as to whether operative work should be done. If it can be done, it is certainly very worthwhile.

ONE other question, in conjunction with numerous problems that could be covered, was this: I remember when I started doing any operating work, I was with Dr. Hetzrot. Dr. Jackson, in his work, was one of the early pioneers in the operative work. Dr. Darragh, Dr. Hetzrot and Dr. Flint, in New York, were among the men who did early operative work. I remember then we used to wait two weeks before we operated on any fracture case, and we used internal fixation. We used aluminum plates we made ourselves or we used Lane's original plates.

Today, we know that about the worst time you can pick to put any fixation in the bone is between the end of the second and the beginning of the third week, because that is when the decalcification process is at its maximum. You have the bone the softest it is going to be throughout the history of the fracture; and if you do fix it, you don't dare try to move it. So it is the one time when, if you can possibly avoid operating, you should not operate.

Why was the original delay of two weeks practised? Because the patient theoretically in that two weeks time built up a ring of leukocytes around this irritated area; walled this area off; and thereby acquired a remarkable additional resistance to infection. The only place that wall of leukocytes and increased resistance to infection existed was in the mind of the man who devised the theory. Nobody ever demonstrated it when it came down to actually demonstrating pathology. That is why I point to the distinction between our old conceptions of fracture pathology and our present one.

The process was supposed to be almost static instead of progressing hour by hour. Eight hours make the difference between a normal elastic, extensible muscle and a solid, infiltrated mass of material that can not be stretched but can only be torn or its fibers torn from one another, in order to get length. What you can do before eight hours and what you can do after eight hours are two entirely different things.

The right time to operate on a fracture is in as few minutes after it happened as is possible. Just as the right time to reduce a fracture by any other means is as soon after injury as possible because the pathological processes changing the whole situation have occurred. It gives you a period of eight hours, sometimes more, sometimes less, but an average of eight hours. You can tell whether it is more or less by the intensity of the patient's pathological signs. But, the time to operate on a fracture, if you figure it is an operative case, is *now*.

We cut down our two weeks time to 10 days; then we cut our 10 days time down to a week; then we cut our week down to four days; then we cut our four days down to two days. It took us 20 years to realize that the pathology was complete by eight hours, and it wasn't a question of waiting days—it was a question of waiting hours. If you operate on them before that pathology becomes fixed in the tissues, which is what happens within eight, 10, 12 hours—if you operate on them before that happens, the hemorrhage which is trying to infiltrate the tissues, the exudate, the inflammatory reaction in the tissues, the edema which follows the trauma all largely escape after operation and through your operative wound; and, 48 hours later, the leg is relatively without edema or induration.

Why? Because the pathology is in the slop pail in the operating room instead of inside the patient's leg. That is plain, ordinary horse sense. There isn't any science about that.

The time to get rid of the pathology is before it becomes fixed, and it is fixed anywhere between eight to 12 hours. That is fact; not theory. It can be demonstrated experimentally. Take an experimental animal, which is what we did in testing this thing out first and give it a fracture with soft part trauma. Operate on it and fix it right away. Take a second case and wait 24 hours; then operate and fix it; and 48 hours later, compare the two treatments.

There won't be any doubt in your mind as to whether it makes a difference or not. It makes all the difference in the world. What is more, the thing you have got rid of is the very factor that because of tension in the tissues has most to do with interfering with the lymphatic and tissue fluid circulation, and definitely the results of early operation are humane in addition to its other advantages.

When this story of operating at our hospital first came along, the senior staff did practically all the cases. They had to show the young men coming on how this thing was to be done. They got pretty good results; but, of course, they were pretty good, and admitted it. Gradually, this conception of the pathological picture grew up, as worked out experimentally. We couldn't laugh it off, and we had to try it clinically.

That meant that instead of the cases being scheduled for two days from now at 9:00 o'clock, they were done at 11:00 o'clock at night, 2:00 o'clock in the morning, whenever they happened. So, the younger men on the staff did them. And, at the end of two years, in evaluating our results, we made the rather embarrassing discovery that the results obtained in the two years at the hands of the younger men, less experienced men, were better than the results that the seniors had secured. So it wasn't a question of who did it. It was a question of less experienced men doing it, getting better results. The only element that fitted into the whole story was the time element—the difference between operating two or three days afterward and operating immediately afterward. It is a tremendous factor.

THERE is one closing point I would like to make that the doctor brought out this afternoon in talking about hips. I noticed a part of his presentation which depicted, from a well known anatomy, perfectly marvelous circulation in the neck of the femur when it is intact.

To make that illustration applicable to the talk which accompanied it, one should visualize a fracture across the whole neck of the femur, which breaks not only the bone but presumably, at least, damages a large part of the circulation which is proceeding from the base of the neck upwards.

The problem of normal circulation of the neck and the problem of circulation to the head after injury are two very different problems. I agree that the coxa valga story in the neck of the femur fractures is unquestionably a vital factor in promoting union. I think it is also perfectly true that elimination of shearing strain promotes union. But, I don't believe it does it by any "physiological challenge," whatever that may be. I think it does it for a very specific reason.

Paunel's book is a fascinating book. It is extremely Teutonic in that it takes, roughly, if I am not mistaken, about 248 pages to say that the more oblique the fracture line which runs through the neck of the femur, the greater the shearing strain —which, I think, is absolutely true. This is proved with engineers' figures and most bewildering diagrams, beyond any cavil or doubt.

But, it is assumed then, or at least one is allowed to assume, that the shearing strain, per se, is responsible for non-union. Many of these fractures never slip. They have the shearing strain present, but they are held by the plaster, for instance or by a pin. They don't lose position, but they don't unite.

The assumption is, and the claim is made, that the shearing strain, per se, the abnormal strain, the unphysiological stimulus, affects the osteoblasts, and they don't make any bone.

I have a hunch that what has happened, and there is a good deal of experimental evidence to support it, is that when two surfaces, bony surfaces, are united by new formed connective tissue, which contains capillaries and lymphatic channels, and you subject that tissue to shearing strain, even though your fragments don't slip, the tissue gets thinned out and stretched and avasculated.

Thus there is a circulatory factor indirectly due to the shearing strain, which is removed when that shearing strain ceases, because the stretching of

tissue in the attempt of the fragments to slip ceases. I think it comes down probably to the same story of the persistence of an acid pH due to relatively inefficient circulation in this perfectly normal healing tissue.

The other factor in the neck of the femur which, I think, is a matter of extreme importance, is this: Except by the *post hoc ergo propter hoc* type of reasoning, there is no proof that osteoblasts form bone in adults. Osteoblasts are distinguished from bone cells, which are supposed to be non-productive as far as bone is concerned, by their shape, their format, and their position, which is along the borders of bone trabecula.

Take any connective tissue cell you want. Decide what shape you would like to have it, and one can grow it that way for you. It won't be done by giving the cell any peculiar properties which it did not possess before. It will be the same connective tissue cell, but, in accordance with the density of the media in which it lies, its form can be varied to suit anyone's desires.

What is more, take a drop of water and some specks of dust. Put the dust in the water, and look at it through a microscope. You will find the specks of dust tend to gather around the periphery of the drop of water where the difference in surface tension exists. That is a very characteristic physical phenomenon. Put some oil on the surface of the water, and more dust, and you will find specks of dust around the edge of the oil drop and the water, where there is a difference in surface tension. Drop a little osteoblast dust, if you want—call them connective tissue cells, just for fun—into a medium which consists of new formed connective tissue on the one hand, with a tissue fluid circulation running through it, and osteoid or bone tissue on the other. You will find the cells concentrated at the place where surface tension differences exist, and they will be round instead of long because of surface tension factors. It is perfectly logical; more logical than the idea that because they lie along the trabeculae they form bone.

When an osteoclast lies alongside a bone trabeculum there is a little hole in the bone where it lies. And, the hole being there and the cell being there, the cell must have made the hole. So, it is an osteoclast. This is hardly a tenable line of reasoning. On the other hand, on the basis of acknowledging that we do not know exactly how bone forms, but accepting proved facts that it will form in the human adult—adults, mind you, not children, infants, or embryos—of new-formed connective tissue, calcium, and pH factors, any treatment that will provide these locally for the patient in the status in which they should be present to get bone is good treatment for fracture, and will cut down non-unions.

Discussion:

DR. GEORGE L. APFELBACH (Loyola University, Chicago): I enjoyed Dr. Murray's talk very much. It is unfortunate that he thought from my discussion this afternoon I thought there were osteoblasts jumping all around that naked femur. I fully realize, and in teaching repair of fractures, as far as we know, I have for two or three years emphasized Dr. Murray's teaching about the change of pH being lower in the first early stage of fracture, and that the cell repair must come from the undifferentiated tissue cells and whatever stirs that up. Whatever stirs that up is the biological challenge, which we talk about.

DR. MURRAY: Why not call it the unknown quantity?

DR. APFELBACH: We have to have a name, and we have to have something to go by. I use the name, not too seriously, of biological challenge. There is a remarkable phenomenon in the physiology and life. I don't think anyone in the United States has done more clear thinking on bone repair than Dr. Murray. I have been very much interested in reading his works, and I like to speculate along the lines that Dr. Murray has.

His explanation on bone repair should have been applied in my talk this afternoon, but I didn't want to get into bone repair. That is the simplest way to put it. There is a disturbance through mechanical reasons of the bone repair. Of course, in the neck of the femur we all know there isn't much chance to produce the same thing that is being produced experimentally; that you have done by putting in the bone tissue, or the Swede, who published an article a year ago, in which he puts bone and finds that the calcium and undifferentiated connective tissues being in there and the proper pH, he can produce bone around the graft even.

DR. MURRAY: You can do the same thing in other conditions. For some years we treated all our solitary cysts by carbolizing the cavity, and then packing with plain gauge as an irritant for 10 days to produce growth of granulation tissue, undifferentiated tissue. Before that has become undifferentiated we fill the cavity with di hexose calcium phosphate.

All of them have healed with solid bone, so the calcium, connective tissue, and pH conditions were the factors involved, without any bone being put in at all.

DR. APFELBACH: Of course, in the neck of the femur you haven't got that situation. You haven't got the surrounding, I presume connective tissue, which is only in the bone itself.

DR. MURRAY: You have no surrounding connective tissue. That is your big problem.

DR. APFELBACH: Maybe there is undifferentiated connective tissue in the actual cortex of the fracture.

DR. MURRAY: It has never been demonstrated, but as a result of accident, you get tissue growth.

DR. APFELBACH: Because you are certainly doing it by fixation. So, I don't think, Dr. Murray, you and I ought to stay up until 4:00 o'clock in the morning because we pretty well agree. I was very much interested in the compound fracture. Of course, at the County Hospital, under the circumstances, we close our skin, but we are doing the same thing you are. We are closing them so loosely that I fail to see much swelling or edema following the closure. We could not, out there, leave the wound open, I feel, with the service we have and the protection on so many cases. But, still I don't feel that we are really closing the skin tight.

DR. MURRAY: You are, really. Leaving it open, I think, is an idealization. In closing loosely, you are avoiding, as far as possible, the risk of closure. When a surgeon says closure of the wound, he means closure of the wound, and he doesn't mean what a great many men who say they close their wounds actually do, which is just very loosely pack them together. That really isn't closure.

DR. APFELBACH: We really get serous drainage. Of course, when you do an open operation and put metallic fixation, you close it then?

DR. MURRAY: Not compound. We do in fresh cases.

DR. APFELBACH: You are making it compound: operating it, putting it in fixation, and still you close it. You don't close it tight there, do you?

DR. MURRAY: We close it fairly tight.

DR. APFELBACH: There is one thing that occurs in a compound fracture that you didn't mention. We all agreed on the early debridement and early treatment just as you stated in your talk. Of course, I think you ought to emphasize the fact that after this is done, you create just as much trauma by trying to manipulate that frac-

ture; that it should never be manipulated after the first treatment. We find if we manipulate a fracture after the second or third day, temperature shoots up. You are producing exudate, trauma, blood clot, and making new open spaces.

DR. MURRAY: Whatever you are going to do, you ought to do that at the outset, and then stay away.

DR. JAMES A. JACKSON (Madison, Wisconsin): I hardly think it is up to any of us to discuss really seriously in any critical manner what Dr. Murray has told us. I would like to say this: This has been one of the happiest evenings I have had since I started the practice of medicine, I mean from a medical standpoint. Many of you, at least some of you, have for years heard me get up and talk on this subject and try to tell the men that you could get good results by open reduction of fractures. But, I have been called everything because I advocated this. I didn't know exactly why I was right, but I did know that I was getting beautiful results in the treatment of fracture by the open method, particularly on the long bones. Then, to sit here tonight and have a scientific argument put forth to show exactly why, has really been a very happy moment to me.

There is one question that I would like to ask Dr. Murray. When he speaks of leaving the compound fractures open, just how does he treat the wound? That is, you leave it practically open, but do you use Dakin's?

DR. MURRAY: No. If we believe a wound is so grossly contaminated that we anticipate infection, that type of wound we will pack open from the beginning and leave it open. We make no attempt to close or introduce sutures. We let it heal by granulation. If, on the other hand, we believe that the wound is such that in our judgment it will probably stay clean, we introduce sutures from within out, leave them untied, but in situ, and then pack the wound loosely with vaseline gauze right down to the bone. If the plate is in there, right down to the plate. We make no attempt to cover the plate. We put a pressure dressing over that. We leave it absolutely untouched for at least five days, with elevation in balanced suspension, post operative. No mobilization during that period because what we are treating right then is infection or the risk of infection, and not fracture.

At the end of five days, we inspect the wound. Here, I think, we have a flaw in our method. If it looks clean to us, when packed with vaseline gauge, we take the latter out—if the wound is pink and red, no edema or any infiltration of the soft spots. If so, we will pack it again, leave it there three or four more days. As soon as the infiltration and edema are gone, we tie the sutures as snugly as we can without creating tension. Frequently, we can't get them completely shut, and we let them granulate in and then pinch graft the area not closed.

I think our error in technic—Dr. Sherman got me on the hip on that, and I had to yell for mercy—is that we do the very thing I am condemning: betting our judgment against the patient's leg as a criterion. I think we ought to take cultures and smears from the wound and close them only if those cultures and smears warrant our closing as dakinized wounds are closed.

I say that seriously because, while I know of only one case that suffered by that closure over a period of 12 years, that one woman lost her leg. That one leg weighs on my conscience because I closed the wound. I don't believe closing a wound without a bacteriological investigation, just on my judgment, was a decent bet for me to make. We inspected, and there was no temperature, no symptoms, everything innocuous. We tied those sutures and I tried to snug it together. I created tension, and within 48 hours she blew up with one of the most violent infections I ever saw. There is a woman who had the same bugs in her after I tied the stitches as she had before I tied them. Within 48 hours she had a violent infection and lost her leg.

It was one of the most dramatic things, as far as I was concerned, because I was deliberately going against principles and creating tension. I think if we use late closure, we also ought in justice to principle to make sure that the bacterial story is such that closure is safe; otherwise, I think, it is just a question of time before we run into a similar case.

DR. E. H. HAMILTON (Chicago): Dr. Murray, I tried out in County to determine pH in fracture, but I was advised that I would have to do it under oil in a very complicated process.

DR. MURRAY: That is only true if you use colorimetric methods.

DR. HAMILTON: Because of being unable to determine the pH, I practically had to leave the problem at that time. You mentioned it was about 4 at the time of fracture.

DR. MURRAY: Within eight hours, it dropped in some cases. The lowest recording we ever had was 4.2, and that is accurate within 0.2 of a pH degree. The average of our cases depends on the amount of tissue damage, of course, the amount of circulatory stasis, and all the factors that cause accumulation of acid products locally. But, the average pH at the fracture site and a few millimeters all around it runs somewhere around, I should say, 6.3 or 6.4 as the average.

DR. HAMILTON: Then you mentioned at the end of 12 days it was up to about 8.

DR. MURRAY: The highest we ever got was a reading of 8.2. The average reading we got at about 12 days post injury ran about, I should say, 7.7, which is above the normal pH.

DR. HAMILTON: The more alkaline, the faster they heal?

DR. MURRAY: That I don't believe we could answer yes or no. I don't know. I will put it this way. I ought to give a positive answer or say no, and we have no evidence that they heal faster. So far as I am concerned, they do not.

The reason I think it is important is this: I think it has a very practical significance. I, myself, believe that if we can control the pH in the tissue fluids at a fracture site, the problem of healing in fractures is solved completely. There can't be any such thing as a non-union unless there is no tissue to ossify. Now, how to control it is another proposition. I have tried all sorts of mechanisms, profusion of the tissues, the use of electrophoresis through the skin, which will only penetrate a millimeter or two, and doesn't get down to the tissues you want to reach.

I know of no method where the pH of the tissue fluids can be controlled continuously with any exactitude. That is, you can improve the condition or you can make it worse, but you can't control the degree to which you do it. The only way we have at hand, the clinical methods we have today, are active motion, massage; in other words, circulatory mechanisms. But, they are not accurate, and we can not control them. If they aren't working, we can't substitute for them adequately.

But, if somebody can ever find a way to control the pH of the tissue fluids and vary them in a given locality, the question of fracture healing is a solved problem, I firmly believe.

DR. APFELBACH: Dr. Murray, do you think that any plate, whether it is steel, with six screws on each side, instead of three, holds a femur in as good fixation as a properly carpentered intermedullary graft well placed?

DR. MURRAY: I think it not only holds it in more rigid fixation, but I think it will hold it in fixation that will allow a function that the graft will not allow. So much so, that when we do grafting procedures, we look on the bone graft as a calcium source for the new formed connective tissues, which is going to form in the region of the graft. We know that if it is moving around, it does what anything else in the tissues moving around does: it creates an inflammatory reaction; you immediately get an acid pH; and you get calcium absorption and not deposition.

When we do it, we put the graft in, and then we plate the femur. The plate holds the femur, and the graft supplies calcium.

DR. GRAHAM KERWEIN (Chicago): I would like to ask Dr. Murray if he does not believe in the function of osteoblasts, how do you explain the fact of the so-called malignant osteoplasia?

DR. MURRAY: I don't. If you think it strange I cannot explain that, I can tell you about 40 other things I can't explain in the question of bone formation. The

reason I say that is this: Of course, the answer to, for instance, an experimental procedure in which you put calcium salts in the presence of new formed undifferentiated connective tissue with proper pH conditions in muscle far removed from bone, (the calcium actually having no derivation from bone), we get bone. Then, as you say, as a result of that procedure, what could an osteoblast have to do with it? There is none in the muscle.

The answer that is always given is that what you have to remember is that connective tissue, skeletal connective tissue, has the faculty of a cell metaplasia to take up this special function of forming bone when the conditions demand it, when the physiological urge is there. That is a grand explanation because nobody could disprove it.

DR. KERWEIN: That is a dual cellular theory, simply.

DR. MURRAY: That is one of the theories of osteoblastic growth. But, in childhood, for instance, you have quite a different problem in all your bone healing and bone growth problems from that of the adult. It is a rather remarkable thing, for instance, in multiple osteochondromas, which are really epiphyseal, which occur in the long bones, and proceed to grow. In the children who have them, if you examine the epiphyseal plate, you find the cartilage to be highly irregular. The cells are irregular in size and shape, and irregular in staining reaction. They look like cartilage out on a big bat. All these osteochondromas in that individual. the cartilaginous portion, will look exactly the same. When the epiphyses close, every one of them changes into perfectly normal bone and stays that way the rest of life.

Something happens at the closing of the epiphyseal lines, whatever is responsible for it, that changes that patient's bone forming mechanism completely. The cartilage all disappears, even in these growths the cartilage disappears, and is replaced by normal bone just as all the rest of his bone is. Whatever happens, whatever the change is, that is what is responsible for the difference between children's healing powers and an adult's healing powers. The correction of deformity, for instance, which a child will do, an adult will not do. More than that, except in cases of terrific trauma with marked gross circulatory damage or in case not of slight, but of very marked interposition of tissues, such as the whole belly of the muscle—with those exceptions it is practically impossible to get a non-union in a child.

With the adult, on the other hand, it is not only not impossible, but it is very easy to get a non-union.

Now, again, to take another phase of that story, and to show you how helpless in a way we are, I have pointed out that rigidity of fixation is very important, empirically, certainly, in fracture healing, in giving at least pseudo-scientific reasons as to why that should be true.

They have a way of treating fractured shafts of the humerus that has been developed in which they put on a plaster cast, which includes the hand and the arm and comes almost up to the fracture line. It doesn't even reach the fracture line, and the arm is allowed to hang at the side, and the patient swings it around, making out of his humerus very much of a star-spangled banner effect, and he gets solid union. I can't laugh that off, and I can't explain it, either.

But, the humerus does heal under those conditions, and there must be some explanation why the humerus will do that, but the tibia will not. You wouldn't dare put a plaster on the foot and up to the fracture line on the tibia, and let the patient wave it around in the breeze. You wouldn't do it to a femur, and you wouldn't do it to both bones in the forearm. But, you can do it to the humerus, and it heals. Why it does heal, I don't know.

But, there is some explanation there. I think these isolated, apparent contraditions can be philosophically accepted at the moment as the exceptions that prove the general rule. Now, all we have to do is to find out why the exceptions. After all, if we knew all about it, all the fun would be gone. We have lots of time to spend trying to work out those problems. But, the question you ask, I can't answer at all.

DR. A. H. WHITTAKER (Detroit): I have enjoyed this talk more than any other talk I think I have ever heard on fracture. I would like to get into the discussion about the metaplasia of the osteoblast. One of the theories of bone formation is known as physiochemical theory. There is a stimulus that takes place at the point of fracture which is in the form of an enzyme, and which has been called phosphatase.

DR. MURRAY: You are not stating that as a fact?

DR. WHITTAKER: No, as a theory. It is rational to believe, however, that when metaplasia takes place, connective tissue also undergoes metaplasia and then differentiates into an osteoblast. Osteoplasia is a so-called ground substance. Nobody has been able to see it under a microscope. Some people think the ground substance is secreted by the osteoblast. With that ground substance present in the region of the phosphatase, the enzyme then is supposed to function and we get bone formed in some distant tissue from the official source. So, that might be one contribution toward the line of thought which would explain the presence of bony tissue in tumor in another part of the body.

Another point I would like to mention. It is fine to hear emphasis on this immobilization of fractures. There is nothing more important to prevent non-union, of course, but Dr. Murray brought out that it doesn't mean immobilization of the extremity. You have to have mobilization of the soft tissue in the extremity, and immobilization of the fracture. At the present time, we have to acknowledge the best way to obtain it is with rigid fixation.

Dr. Murray has also mentioned this thought along the fracture treatment line: that it is not a static thing, but it must progress. I believe, rather than accepting rigid fixation as the final way to solve this problem, we should get this fixation; and at the same time get rid of the metal we are forced to use. We are trying to make a little contribution by doing as the dentist does: forming an inlay, opening the edges, and tightly fitting into those small pieces of bone from the shaft of the bone nearby. Where there is not too much stress on the bone, we get rather good fixation that way.

Another method is making fair-sized holes through the fracture line, taking spicules of bone from a nearby shaft, and driving them tightly into these openings. Of course, it is nothing like steel, but at least it is a step toward fixation by the patient's own tissue, which I believe to be the ideal.

DR. MURRAY: I might say, Dr. Whittaker, that the work of Honor Bridgett Fell, in England, on the question of phosphatase has left everybody with the impression that phosphatase is a ferment peculiar to bone. As a matter of fact, there is as much, if not more, phosphatase in the kidney as in the bone. Phosphatase is found in various regions of the body under certain conditions in marked concentrations.

It is a very tenable idea that phosphatase is not only not produced by anything connected with the bone, but is merely the serum phosphatase which is found in the serum and is concentrated merely in those regions where there is work for phosphatase to do, because of biological conditions. The reason it is in the kidney is because of the excretory function of the kidney and the excretion of salts which the phosphatase works on. So, it is very possible that the presence of the phosphatase in bone really doesn't indicate that the bone itself produces phosphatase.

I think the one argument in favor of its producing phosphatase is this: You take a three-day chick embryo and microdissect out the anlage of the femur and put it into tissue culture. That anlage will grow the general shape and form of the femur even in the artificial medium. It will never produce epiphyseal lines; never undergo any differentiation in the bone forming cartilage; and it will never produce any phosphatase.

If, instead of taking the three-day chick embryo, you take the six-day chick embryo and transplant the femur into artificial medium, it will not only differentiate epiphyseal lines, it will differentiate bone forming cartilage, and will develop phosphatase in the anlage in the artificial medium. That is another one of those things you can't explain.

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FRAUDS in payroll reporting are vicious and costly. Protective measures and internal audit control serve as a vitalizing deterrent in stemming loss of revenue. False classifications, understatement of totals, twin sets of books and innumerable other abuses can be effectively curtailed by the introduction of and strict adherence to standardized payroll forms and audit procedures. The recent fraud investigations revealed that millions of dollars are lost annually to companies. With the invaluable experiences accumulated through these investigations, constructive teaching and instruction will be enthusiastically absorbed by all payroll auditors. Comparisons with Federal Social Security and Unemployment Insurance reports are extremely helpful in curtailing fraudulent activities. Close the avenues of fraud and premium income, unaffected by claims, becomes surplus funds.

Only Compulsory Industrial Insurance

IN THE memorable year of 1914, society witnessed the initial momentous social advance in the United States; the introduction of workmen's compensation insurance as the obligation of industry. From then to now, history has recorded the beneficent economic and scientific progression of this evolutionary legislation. It is striking to note that in New York State, workmen's compensation insurance is the only form of compulsory industrial insurance underwritten by private and mutual carriers, every other type is voluntary (unemployment insurance excepted.) This varied underwriting tends to establish efficient and keen management, minimum rates, competitive service and wholesome productivity. The State Insurance Fund, an organization created and supervised by the legislature, maintains a premium income of approximately 25% of the total yearly revenue.

Relation of Medical Cost to Premium Income

THE doctor is the pulse of the insurance carriers' success. With the energetic cooperation of the medical profession the carriers prosper, without it there is resulting disorganization. Twenty cents of every premium dollar is expended for medical care. Every injured employee requires the services of a physician; no award adjudicated without medical evidence. The doctor, in thousands of cases, is the first reporter of an injury. The quality of medical care determines length and extent of disability. What more pungent testimony is there to the intrinsic need of harmonious relation between the members of the medical profession and the insurance carriers? The doctor's worth must be appraised in that vein.

For the first time since the enactment of the law, the legislature, in the year 1935, granted the injured employee the right of free choice of physician. Eighteen thousand doctors enrolled in the movement. The revision was a consequence of uncontrolled medical abuses which infested the administration of workmen's compensation, such as mass production medical clinics and unscrupulous solicitation. The expedient invoked by many companies in providing the most competent medical and surgical care was socially and economically praiseworthy and could not be attacked in that industry and labor were the beneficiaries. However, the indiscreet activities of a minority group brought public contempt and corrective legislation.

What have the experiences in medical and disability costs been from 1935 to date? The trend in expense per case is consistently rising. Why? Is the claimant receiving a higher degree of medical care? Or is it that free choice of physician relieves the carrier of control?

To accurately define the many confusing issues which have distraught the insurance carrier and the doctor will evoke mixed comment and criticism. But, it can be stated, unequivocally and forcibly, that the conduct of a few insurance carriers needs modification, disciplinary measures must be adopted and exercised by the various county medical societies and finally, fundamental

changes of the existing law should be acted upon.

How Medical Costs Can Be Reduced

FREE choice of physician has revitalized the interest of the members of the medical profession in the treatment of injured workers. Section 13 A of the Workmen's Compensation Act, specifies clearly and comprehensively, the means by which the claimant sets into motion the legal obligations of the doctor and the insurance carrier. For a greater part of the medical and surgical procedures, written authority to the physician is required. Yet, some carriers are making it increasingly difficult for the qualified doctor to obtain that necessary authority, with the disquieting result that many sincere and conscientious physicians are being driven from the field of traumatic medicine. What effect on medical and disability costs?

The confused claimant is compelled to trot from one doctor to another, oftimes receiving identical treatment which prolongs disability, increases medical costs, infuriates the doctor and ultimately, dissatisfies and disgruntles the claimant who then is determined on an avengeful course. Could not the carriers issue authorization with dispatch, thus permitting the doctors to undertake and continue treatment, thereby reducing the probability of extended care and incapacity. Authorization for hospitalization has, in many instances, been delayed and refused. The claimant, disabled as he is, in need of essential hospital care, either remains at home or travels about in search of assistance only to aggravate his injury. Contesting the righteous case because of principle rather than for the encouragement of sound public relations, adds not alone to unnecessary administrative costs, but to medical and disability expenditures.

The harmful practice of lay representatives questioning the medical wisdom of the doctor stirs ill feeling and hatred. When a valid dispute arises as to the course of medical or surgical treatment, it would be the wiser course for the carriers' doctor to make inquiry, examine the claimant and then collaborate with his physician. Medicine is spoken through the mouths of doctors, none others.

Let there be a cessation of the uneconomic campaign of hostility; the law governs our activities and the best interests of the injured employee demands prompt medical attention, coherent administration and a more fervent devotion to the fundamentals of idealistic association. To ignore the living fact that efficient medical care reduces both medical and disability costs and galvanizes relations with the claimant is to lose sight of one of the predominant principles of competent claims management.

The Obligations of the Medical Profession

WHEN the free choice amendment came into being, the medical profession assumed a profound and deep obligation to society. The doctors responsibility is not alone to the patient but also to the State. Accusations have been directed at the Medical Societies for their failure in carrying out the provisions of the Workmen's Compensation Act, as enumerated in section 13-d. The medical societies should look upon injured workers as their wards, and shield them from abuses by unqualified practitioners and from unethical and unprofessional tactics. Medicine should coordinate its duties and functions with those of the insurance carriers. Closer cooperation and a firmer understanding will lead to smoother administration to the distinct advantage of the doctor and claimant.

Recommendations for Amendments to the Law

THE law makes provision for arbitration of medical bills, in fact the sections are obligatory. The system is unsound. The carriers have been misguided in the belief that arbitration has resulted in lowering medical costs, when in fact, too much of the reverse has been true. Only a fraction of the total number of medical bills find their way to the board of arbitration. The threat of arbitration to a doctor acts as a gentle coercive gesture. What is the doctor to do? Submit to arbitration or consent to a voluntary reduction of his charges? He consents to deductions against his better judgment. Arbitration involves not alone delay in collection but valuable time lost in appearance before the board. The much publicized fact that bills submitted to arbitration have been cut approximately 33⅓% is another fearful consideration. But the doctors do not forget and only doctors treat injured employees. In no other industry or business is there the element of contingency attached to the collection of fees for services rendered. The doctor is compelled to await the outcome of the compensibility of the case before the Labor Department, and in many cases, months expire before a final determination is made. In the event of a disallowance of the case because of a legal technicality, the doctor is barred from collection and must resort to the claimant, who more times than not, is financially irresponsible.

As a substitute for arbitration, it is suggested that a division for the consideration of medical bills be created in the labor department, supervised by Medical Referees. Only qualified members of the medical profession should be appointed, on a full time salary. In the interests of expediency, efficiency, equity and with an eye toward the reduction of unnecessary medical cost, this proposed change should interest both the doctor and insurance carrier.

In addition, the section regulating the choice of physicians should be altered to permit the examination coincident with or shortly after the occurrence of an injury by the doctors employed by the Labor Department to determine the extent of injury and to further permit the employer or carrier to recommend, with the assistance of the Medical Society, specialists or surgeons as the case may require. This step will eliminate duplication of treatment, provide for proper care immediately, afford the Labor Department greater facility for

the determination of awards and eliminate costly abuses existent under the present law.

Conclusion

THE interests of the insurance carrier, the doctor, and the claimant are parallel in their duties to industry, which provides and pays for this insurance and to the State which superivses and directs the administration of the act. A deeper appreciation by the insurance carrier of the economic and sociological burdens of the injured worker, unable to care for his family and by the physician of the far reaching statutory duties imposed on the insurance carrier, will contribute firmly to the evolution of an acceptable and forceful system of workmen's compensation insurance.

Gall-Bladder Disease

— *A Consideration of Mortality* —

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GALL-BLADDER disease is probably the most common intra-abdominal condition and manifests itself in a variety of ways. It is almost uniformly associated with symptoms which vary in degree, in disability, in danger and in the production of distant or remote sequelae. It is the general opinion that the gall-bladder functions as a reservoir after the concentration of bile and as the control of the ebb and flow of bile into the intestinal tract. Infection of some type is basically the etiological factor in the production of pathological changes in the gall-bladder, extra biliary system, the liver, pancreas, and more remote organs. No age or sex or constitutional status, is immune from gall-bladder affections nor wholly protected from its effects.

The main symptoms of gall-bladder disease are primarily local and confined to the gastro-intestinal system. There are, however, certain allergic states that are due to a low grade, continuous infection of the gall-bladder. There are, in addition, undoubted cases of remote effects in the production of various types of arthritis. Again, there is a systemic effect of gall-bladder disease as manifested in continuous ill-health, called "cholecystic toxemia." Furthermore, even a moderate experience in gall-bladder symptomatology will discover not a few cases of cardiac disturbance that are basically the result of gall-bladder disease. The latter statement is not intended to include the mistake or error in diagnosis that is occasionally made in considering a case of coronary thrombosis an acute gall-bladder manifestation or as more frequently obtains, an atypical gall-bladder attack simulating the syndrome of coronary disease.

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From January, 1920, to June, 1937, 5000 consecutive cases of gall-bladder disease were analyzed by DRS. HEYD, CARTER and HOTZ. Of these 3986 were operated upon by 53 different surgeons; 50% of the number of the operated cases were private and 50% were so-called clinic patients. In these 3986 operated cases there were 309 deaths from any cause whatsoever, giving a basic hospital gall-bladder mortality of 7.7%. (Table I.) This general figure of 7 to 8% mortality is in general

TABLE I.

ANALYSIS OF 3,986 CONSECUTIVE OPERATIONS ON THE BILIARY TRACT (1920-1937)

Operation	Total Number	Mortality (No.)	Mortality (Per Cent)
Chronic Cholecystitis			
Cholecystectomy	3,240	190	5.8
Alone or with appendectomy	2,438	88	3.61
With dochostomy	238	27	11.34
With other operation	568	74	13.03
Cholecystectomy with dochostomy and other operations	6	1	16.16
Cholecystostomy	66	22	33.3
Alone or with appendectomy	43	13	30.24
With dochostomy	16	6	37.50
With other operation	7	3	42.80
Total	3,306	212	6.40
Obstructive Biliary Disease			
Cholecystogastrostomy	52	15	28.8
Choledochostomy only	37	13	35.1
Choledochostomy with other operations	2	2	100.0
Plastic on ducts	5	3	60.0
Total obstructive	96	33	34.4
Acute Cholecystitis			
Cholecystectomy	517	45	8.82
Alone or with appendectomy	428	32	7.47
With dochostomy	89	13	14.60
Cholecystostomy	45	13	28.80
With dochostomy	9	3	33.33
Total cholecystostomy (with 3 other operations)	57	18	29.63
Total acute cholecystitis	574	64	10.97
Total for all biliary tract operations	3,986	309	7.7

accord with other reports covering a like period of time and approaching it in numbers. In the "break up" of this operative material a number of significant facts emerged. The clinical and mortuary impact of these statistics is arresting and resulted in some significant deductions in regard to diagnosis, pre-operative treatment, operative technique, postoperative treatment and the eventual curability and morbidity of the surviving patients.

SERIATIM, we may consider a few of the most outstanding features of the study:

1. THE CONTINUOUS AND PROGRESSIVE CHARACTER OF ALL INFECTIONS OF THE GALL-BLADDER. It became apparent that disease of the gall-bladder does not spontaneously "cure" itself but from the very nature of the disease is a continuous, pathological process with a high degree of cyclic periodicity in symptoms. This inherently progressive course

of gall-bladder disease involves in chronological order the gall-bladder, cystic duct, the common duct, the liver, the pancreas, and finally affects the entire organism. This pattern of continuous and progressive change varied, to be sure, but in the main the evolution of symptoms was dependent upon chronicity, the constitutional habitus of the individual and the inherent degree of infectivity, plus the mechanical factor of obstruction in the ducts from either inflammation or calculous formation. With chronicity, the number and severity of the symptoms increased, the technical difficulties of surgical intervention became greater, and the mortality rate invariably rose. For example, there were 1270 operations on patients with symptoms of chronic cholecystitis of less than two years' duration. Nine hundred and fifty-nine of these operations were uncomplicated, with 13 deaths, or a mortality of 1.38%. However, in the same group there were 311 patients with complications, individuals who had a history of a previous acute attack or the gall-bladder surgery was complicated by some additional surgical procedure. Twenty-two of these patients died, giving a mortality of 7.10%. (Table II.)

TABLE II.

AN ANALYSIS OF THE RESULTS OF OPERATION IN CHRONIC CHOLECYSTITIS WHEN DEFINITE SYMPTOMS HAVE BEEN PRESENT LESS THAN TWO YEARS

	No.	Deaths	Per Cent Mortality
Uncomplicated cases	959	13	1.35
Complicated*	311	22	7.10
Totals operations	1,270	35	2.75

Causes of Death		Major Complications	
Pneumonia	11	Wound infections (severe)	23
Peritonitis	10	Dehiscence	15
Liver death	4	Pneumonia	8
Cardiac failure	4	Thrombophlebitis	5
Operative shock	3	Postoperative hemorrhage	4
Postoperative hemorrhage	2	Pleurisy (effusion)	3
Uremia	1	Cardiac failure	3
		Surgical erysipelas	1
		Peritonitis	1
		Acute parotitis	1

* Those with previous acute attacks or with secondary operations.

It is a generally-held opinion that stones in the common duct are sequential to calculi in the gall-bladder. Stones in the common duct were found in 6% without any calculi being present in the gall-bladder. It is, however, easy to conceive that an infection of the gall-bladder may, by reason of mucosal conditions, not produce gall-stones within the gall-bladder, but by constant bathing of the common duct with infectious material produce "de novo" calculi in the common duct.

Choledochostomy was performed simultaneously with cholecystectomy or cholecystostomy for chronic gall-bladder disease in 260 patients, and calculi in the common duct were found in 82% of these patients, and in 3306 operations for chronic cholecystitis the common duct was explored in 7.7%. According to the report of the pathologist associated "pathological changes in the common duct were almost invariably associated with severe pathological changes in the gall-bladder." The statement validates the assumption that common duct disease is usually secondary to gall-bladder disease. Symptoms of gall-bladder disease in patients with common duct calculi provide a rather interesting observation as to the duration of symptoms and mortality. There were 1270 patients with symptoms of gall-bladder disease of less than two years. Twenty-four had stones in the common duct, representing a percentage of common duct stones of 1.9. In other words, in any group of individuals with gall-bladder symptoms of approximately two years' duration at least, 2% will have common duct stones. There were 1020 patients with symptoms of gall-bladder disease from two to 10 years. Ninety-two had common duct stones and the percentage of common duct stones rose to nine. In 610 patients with gall-bladder symptoms of from 10 to 35 years, 97 had common duct stones and the percentage of common duct stones rose to 16. (Table III.)

TABLE III.

DURATION OF CHOLECYSTIC SYMPTOMS IN COMMON DUCT STONE

Duration of Symptoms	Total Cases	Common Duct No. Cases	Per Cent Common Duct Stone
Under 2 years	1,270	24	1.9
2-10 years	1,020	92	9.0
10-35 years	610	97	16.0

It is interesting that in 574 operations for acute cholecystitis—the diagnosis of acute cholecystitis was entertained only when made by the pathologist—80 patients had common duct stones, an incidence of approximately 14%, and in only three of these patients was there lacking a history of previous gall-bladder symptoms. In other words, in the acute gall-bladder cases approximately 96% had a definite history of chronic gall-bladder disease and 80% of all the secondary gall-bladder operations followed a cholecystectomy.

2. MULTIPLE SURGERY. About 13% of the operations in women with gall-bladder disease were associated with fibroids and nearly 10% of all the series, both male and female, had gastro-duodenal ulceration.

It is a rule at the Post-Graduate Hospital to remove the appendix in practically all intra-abdominal operations. The mortality of operations on the gall-bladder, with or without appendectomy, showed practically no variation between the two groups. However, when other operations were added to cholecystectomy, the mortality increased rapidly with each type of secondary operation. If there were added to any gall-bladder surgery a gastro-enterostomy the mortality became 16.4%; with gastric resection, the mortality rose to 31.1, and with hysterectomy a mortality of 11.8% was obtained. It is obvious that surgical intervention for gall-bladder disease is an opera-

tive intervention of sufficient magnitude to be the sole and only indication for the surgical intervention. (Table IV.)

TABLE IV.

CHRONIC CHOLECYSTITIS

Factors Influencing the Morbidity and Mortality in Surgery for Chronic Cholecystitis

	No. of Cases	Morbidity	Per Cent Mortality
(A) Multiple surgery (Cholecystectomy plus secondary operation	575	1.72	13.85
1. With gastroenterostomy	128	.....	16.4
2. With pyloroplasty	111	.....	9.9
3. With gastric resection	61	.....	31.1
4. With acute appendix	31	.....	13.0
5. With hysterectomy	59	.....	11.8
(B) Conservative treatment in acute cholecystitis			
1. Subsided acute	474	1.59	11.02
2. With chronic abscess or old perforation into colon	46	2.20	38.0
3. Cholecystostomy in previous acute	15	1.58	20.0
(C) Cholecystostomy in chronic*	68	1.50	7.4
(D) Jaundice (especially necessitating common duct surgery)	254	.....	13.0
(E) Delay in surgery for chronic cholecystic symptoms. Series with less than two years' history	959	.....	1.35
Total series of chronic cholecystitis	3,303	1.31	6.4

* Means cholecystostomy for a previous chronic infection—now followed by a cholecystectomy.

Earlier, I referred to the fact that in this large series 7.7% of the patients had the common duct explored and that stones were found in 82% of the cases, with a mortality of 11.34. I also stated that four-fifths of secondary operations on the common duct were in patients who had had a cholecystectomy. Choledochostomy following a previous cholecystectomy was done in this series in 39 instances, with a mortality of 40%. Only six of these 39 patients had their original operation at the Post-Graduate Hospital: 33 were done in other hospitals. Of the 39 patients who had common duct operations following a previous cholecystectomy 32 of the group, or 80%, had recurrent overlooked stones. The inference is obvious, that any operation for gall-bladder disease should, if and when there is evidence of common duct disease, be completed by exploration of the common duct as part of a single operative procedure.

Considerable controversial gall-bladder literature has arisen in the discussion of acute cholecystitis. Like most discussions more heat has been generated than light. We are not inclined to believe that there is any such "immediacy" for surgical intervention in acute gall-bladder disease as applies to gastro-intestinal perforations or acute appendicitis. In other words, the term "immediate operation" as opposed to "deferred operation" is a misnomer. Acute cholecystitis — the diagnosis being made only by the pathologist—occurred in 14.5% of the series, whereas the incidence of stones in the gall-bladder in chronic cholecystitis was approximately 70%; 89% of the acute cases had calculi in the gall-bladder. Undoubtedly the mechanical factor of calculus is a factor of marked importance in initiating the acute phases of cholecystitis. It has been found that the individual with an acute gall-bladder who had a hospital residence of from six to 24 hours before operation produced as a group the least mortality in this condition. One hundred and twenty-eight patients were operated within six hours after their admission into the hospital, with a mortality of 16.6%, and this is almost exactly the same mortality of 80 patients whose disease began without 48 hours of their operation, namely 15.10%. Two hundred and ninety-seven patients were prepared pre-operatively from six to 24 hours, with a mortality of 7.4%, or within the basic mortality of the entire group 7.7. One hundred and forty-nine other patients were prepared from two days to 24 days, with a mortality of about 13%. Of 316 cases observed 16 hours or longer 57% had a continuous pgoression of their symptoms and physical findings. In only 12% was there definitely a subsidence of the clinical picture.

Jaundice is rightly believed to indicate chronicity of disease: of the 574 acute cases a definite history of jaundice was present in 101, with a 15.8% mortality and jaundice was present at the time of operation in 155 patients, with a 20% mortality. In other words, the history of jaundice increases the mortality expectation over 100% and jaundice at the time of operation increases the mortality expectation 200%. (Table V.)

TABLE V.

ACUTE CHOLECYSTITIS

Factors influencing the mortality and the morbidity in surgery in Acute Cholecystitis

Factors	No. of Cases	Mortality
B. Degree of Pathology (Attending Pathologist's Diagnosis)		
(1) Acute Cholecystitis	206	5.85%
(2) Purulent cholecystitis	117	9.40%
(3) Gangrenous cholecystitis	150	7.33%
(4) Perforated, with abscess	16	0.00
(5) Perforated, with peritonitis*	51	35.85%
* Exclusive of 16 cases with clinical peritonitis where cholecystostomy was done.		
B. Complicated Cholecystitis (Comparison with Acute only)		
(1) Acute only	206	5.85%
(2) Complicated cholecystitis	368	14.10%
(3) Perforated cholecystitis (known)	69	26.10%
C. Duration of Present Illness		
(1) Operated within 48 hours of onset	81	11.10%
(2) Between 48-96 hours (2-4 days)	137	5.90%
(3) Between 4-7 days	104	7.70%
(4) Between 7-50 days	182	16.71%
D. Duration of the Period of Hospital Observation Pre-Operative		
(1) 0-6 hours	128	15.62%
(2) 6-24 hours	297	7.41%
(3) 24-48 hours	58	10.35%
(4) 2-24 days	91	17.59%
E. Jaundice		
(1) Definite history of jaundice (past)	101	15.80%
(2) Jaundice at time of surgery	155	20.60%

Finally, the cyclic character of gall-bladder disease is indicated in a consideration of Table VI. The pathologist divided his material from the 574 cases of so-called acute cholecystitis into acute, purulent, gangrenous or perforated. The mortality in the first three of these categories, "following a single acute attack, with no definite history of a previous cholecystitis," is well within the mortality rates of simple, uncomplicated cholecystectomy, namely 3 to 5%. However, in 401 cases of acute cholecystitis (in 34 similar cases no pathological tissue was obtained) with a history of one or more acute attacks or a definite history of previous chronic cholecystitis, the operative mortality was approximated double in each of the categories—acute, purulent and gangrenous cholecystitis.

TABLE VI.
THE CYCLE OF ACUTE GALL-BLADDER DISEASE
(as seen in 574 pathologically acute gall-bladders)

Clinical Course / Pathological Equivalent	Single Acute Attack—No Definite History of Previous Cholecystitis			
	Acute	Purulent	Gangrenous	Perforated
No. of cases in each group..	51	25	42	23
Mortality per cent	3.9%	4.0%	4.8%	26 %
Clinical Course	**History of One or More Acute Attacks or a History of Chronic Cholecystitis**			
Equivalent Pathological	**Chronic Acute or**	**Purulent**	**Gangrenous**	**Perforated**
No. of cases in each group..	155	92	108	46
Mortality per cent	6.4%	10.7%	8.3%	33.7%

Traumatic Injuries and Mental Disturbances

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THE casual relationship between certain occupational diseases and psycopathic mainfestations, is well established in intoxications by lead, certain hydrocarbons, carbon disulphide, manganese and carbon monoxide. Fatigue and caisson disease are often also responsible. There is, however, a definite controversy over the relation of head injuries and subsequent mental disturbances. Not infrequently, a mental syndrome develops within a year of the occurrence of a head accidental injury. There appear cases of mental alienation with delirium, a state of excitation, depression, rapid diminution of mental faculties, fixed ideas, disorientation and even death. This may be preceded by a change of personality, restlessness and mental confusion which necessitates confinement to a sanatorium.

In the determination of the occupational factor, its true relationship to employment must be established. Alcoholism must be ruled out and any other pre-existing mental condition or predisposition should be carefully analyzed.

Ordinarily a violence to the physical structure of the body particularly the head, will bring out a psychopathic status that is latent. Latency and predisposition are often ignored by compensation courts if a mental state follows an injury in the course of employment.

When the clinical symptoms of a mental disturbance appear five or more years after the occurrence of an accident, the relationship is very difficult to establish. The functional disturbances usually produced by head trauma such as buzzing in the ear, neurasthenia, headaches and mental confusion, produce such inquietude which eventually leads to some form of mental disorder. It is logical to assume that had there been no accident, possibly, no mental disturbance would have resulted. Nevertheless, heredity and the constitutional factors related to the cortical cells and endocrine disturbances which are extracerebral, disturbances of sexual functions and other forms of autointoxication should be carefully weighed.

It is not sufficient to postulate, but every case should be argued and properly evaluated. Traumatic psychosis may be presumed:

1. If it follows immediately a trauma and is characterized by irritability and hypocondria;
2. If any other preceding or intercurrent cause such as infectious diseases can be excluded;
3. If the injury has been limited to the head and is of a serious nature;
4. If the psychosis has developed within a period of three to five years.

Epilepsy and apoplexy together with disturbances of sensation or paralysis of motor nerves, constant headache, change of personality and irritability of a progressive nature, and inability to tolerate alcohol or excessive smoking following a recent or distant head trauma, may be ascribed to traumatism.

The usual diseases encountered as an aftermath of head injuries are, dementia precox, paranoia and the general paralysis of the insane. The latter being more frequently ascribed to nervous syphilis. The disability induced in terms of loss of earning power should be based on the severity of the psychic symptoms and the relative prognosis:

1. Chronic psychic weakness involves loss of wage earning capacity from 60% to 100%.
2. A post-traumatic confusional state which is permanent, entails a loss of earning capacity from 30% to 75%.
3. Traumatic melancholia should be awarded from 20% to 50% disability.
4. Dementia precox, paranoia and dementia paralytica involves a 100% total disability.

CLINICAL CASES: The following cases are illustrative of trauma and mental diseases:

CASE No. 1—W.W., age 31, male, white, received in Hospital X on May 26, 1940. He gave a history at that time of having been in an automobile accident 20 hours previously at which time he was knocked unconscious. He complained of headache, and examination was essentially

negative. He was sent home to bed and advised to return if further symptoms developed. He came back the next morning complaining of marked dizziness, headache and vomiting and was admitted to the surgical ward with a diagnosis of "cerebral concussion." Physical examination and neurologic examination was still negative except that the tongue appeared to protrude to the left side. Blood pressure was 170/90. He was kept in the hospital until June 1, 1940. His headaches disappeared the last two days in the hospital and he had no vomiting.

He had two spinal punctures, the first one on May 27 at which time the initial pressure was 20 cm. of water and dropped to 13 cm. of water with the withdrawal of 8 cc. Fluid was clear and colorless. The second tap was made on May 30 at which time the initial pressure was 14.5 cm. of water and dropped to 10 cm. of water with the withdrawal of 1 cc. Fluid was again clear and colorless.

The day after discharge, the headache returned and the patient was seen several times at the receiving ward and in the Surgical and Neurologic Clinics. Finally on June 18, 1940, he gave a story that he had become quite nervous and without realizing it, had been threatening his wife with the butcher knife. He was received in the hospital for further study.

While in the hospital, the patient's symptoms recurred at regular intervals. The eye grounds and visual fields were normal. The deviation of the tongue to the left was explained to be due to a disturbance of the right temporomandibular joint. The findings were insufficient to make a diagnosis of an organic lesion. It was felt that the patient's symptoms were entirely due to a post-traumatic psychoneurosis.

Case No. 2—W.G., age 20, male, white, admitted to Hospital Y in August, 1939, at which time he was knocked unconscious and treated for a head injury as the result of a street car accident. He remained in the hospital two weeks. He was apparently well and was told to return for a Barany test. His mother, however, noticed that he had difficulty in walking, and veered to the right. He returned to the hospital five weeks after, for further study. While there, he apparently became psychotic and fought several times with physicians and nurses. The family and past history, were negative.

The mental examination as to attitude and behavior, disclosed a tendency to rambling and locquaciousness. He kept repeating: "I don't think I'm crazy, I'm just nervous. You can't blame me after I had a head injury. The bad tasting medicines have a bad effect on me." He showed no delusions or hallucinations. The sensorium was clear. He further stated that he must be out of his mind at the hospital because he never tried to hurt anybody.

The diagnosis in this case was made as:

1. Manic depressive psychosis.
2. Post-traumatic psychosis.

He was discharged from the hospital two weeks after admission in an improved condition.

Case No. 3—W.S., age 55, male, colored, admitted in Hospital X on September, 1940. He complained of "heat spells" consisting of vertigo, headache, sweating and frequent fainting attacks. He gave a history of having fallen while going down stairs 10 days previously, incurring a laceration of the scalp which was subsequently infected. He was very uncooperative, belligerent and unruly and tried to escape from the ward. He has become markedly hallucinated.

The diagnosis was: Post-traumatic personality disorder He was discharged 10 days after admission in an improved condition.

Case No. 4—C.C., age 16, male, white, admitted in Hospital Z on October 26, 1936 or four years previous to admission in hospital W. The history given was that four years previously he was injured in the head while riding a bicycle which ran into an automobile. The x-ray report, and examination showed that he had an oblique linear fracture of the left side starting from the left parietal eminence and passing downward and forward to the middle central fossa with no evidence of depression. He had been discharged in good condition and with no neurologic signs. Four years later the school authorities noticed that he was suffering from a post-traumatic behavior change and his discharge with supervision was recommended. When admitted to hospital W on May 7, 1940, it was disclosed that he had been a good student prior to the accident but since then had not been able to concentrate. He hated to attend school and was absent most of the time. Recently, he had joined a company of boys and had robbed four houses for which he was apprehended by the police.

He complained of severe headaches and dizzy spells, but no convulsions or unconscious periods. There was a distinct personality change following the head injury. The encephalogram and general neurological examinations were negative. His first delinquent history such as house breaking and burglary, began four years after the skull was fractured.

Case No. 5—A.B., age 54, male, white, admitted to Hospital W September 21, 1940, and discharged November 6, 1940. A history gleaned from various sources, revealed that in 1907 he was struck on the head with a heavy piece of iron thrown by a fellow employee while at work. Recovery at that time was uneventful. Ten years later he fell and struck his head on the concrete floor while employed as a baggage hand in a railroad station. He recovered but was subject to severe headaches at frequent intervals, following this injury. Four years later, he sustained an injury in his back and soon after, was committed to a state hospital where the final diagnosis was that of paranoia. He came to hospital W because he found out that lately he has not known what he was doing. He stated that he was "slipping and was not willing to do anything neither did he know what he was doing." The diagnosis was made as a depressed phase of manic—depressive psychosis. An indirect left inguinal hernia was found and corrected and the patient discharged as improved.

Case No. 6—A.A., age 42, male, white, admitted to Hospital W on August 31, 1937, and discharged March 18, 1938. He was referred to the hospital after being arrested charged with breach of peace, in that he was preaching to a number of pedestrians in a loud and boisterous voice. He was excited and had marked hallucinations in the religious field. He stated that he had received a message to go forth and preach. He had a tendency to violence. He also had suicidal ideas and threatened homicide.

The history revealed that in 1932, or five years prior to admission, he had a traumatic injury on the left side of the head, incurred while working in the mines. He then developed a dimness of vision and associated headaches which had been present at intervals. The headaches increased in frequency and severity with the onset of his visual field limitation.

The final diagnosis at the hospital was schezophrenia and post-traumatic psychosis with mental deterioration.

Case No. 7—T.T., age 47, male, white, admitted to Hospital X February 24, 1938, and discharged April 5, 1938. The present condition began in 1931 when he suffered a skull fracture in a hold-up. A plate was inserted at that time. On admission, he had scratches on the forehead and it was learned that he had fallen the night before. He had become unconscious and had spasms.

During his stay at the hospital, he had a number of clonic convulsions but did not seem paralyzed on any side. At times he was very emotional, crying continuously.

The final diagnosis was, old cerebral injury with damage to left frontal lobe and traumatic epilepsy.

Case No. 8—R.B., age 30, female, colored, admitted to Hospital W May 5, 1936, discharged July 10, 1936. The patient had been admitted the previous month in another hospital suffering from a basal skull fracture with bleeding from the right ear and alcoholism. Her course in the hospital had been downward. At first there were lucid

intervals supplemented by maniacal episodes. She had always been disoriented, uncooperative and irrational.

The diagnosis was that of traumatic psychosis. She had a positive Wassermann. The mental examination revealed that she was mumbling incoherently concerning the death of a brother. At times she said she suffered from delusions saying that someone was trying to shoot her. Her orientation was bad and memory almost gone.

Final diagnosis was, traumatic psychosis, chronic alcoholism and lues. At discharge she was improved.

Evaluation of Manipulative Therapy in Back Conditions

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IN ORDER to evaluate the manipulative therapy of low back conditions, the author has presented various types of low back lesions and classified them according to their response to this mode of treatment. In a previous paper, there was outlined the particular group which respond in the most favorable manner. That enumeration will not be repeated at this time. However, the point is insisted upon that in order for any low back condition to be treated successfully, an accurate diagnosis must be made. It is only by this means that treatment can be applied intelligently rather than empirically and that the patient can obtain relief and return to his normal activities at the earliest date.

GROUP I. *Conditions responding to manipulation with immediate relief and correction.*

Cases of acute sacro-iliac and lumbosacral strains respond most satisfactorily to manipulative treatment. Generally the diagnosis is strongly suggested by the history, the traumatic incident is definite, very often there have been no previous episodes of low back pain, and the clinical picture is quite characteristic: since the onset of pain the patient may have been locked in a position of forward flexion and lateral inclination or list, accompanied by severe pain in the low back perhaps radiating in an acute manner down the thigh. Localized tenderness is situated over the affected joint, whether lumbosacral or sacro-iliac.

Illustration: A medical student in his junior year came in with the complaint of acute backache. He was somewhat better than medium height, of good muscular development and apparently in good physical condition in spite of his present incapacitating plight. The previous afternoon, according to his history, in beginning to chop some wood, he was suddenly seized with a sharp pain in his low back just as he went through the rotary flexion movement of swinging the axe downward. He was immediately unable to completely extend his spine again and had since assumed the position of marked listing to the left. Shortly after the onset, he was conscious of pain radiating down his right thigh along the distribution of the sciatic nerve. Following the removal of his clothing, an appraisal of his standing position posteriorly emphasized the list of the trunk to the left with the right paravertebral muscles visibly taut. The left shoulder was higher than the right. The right posterior superior spine was slightly higher than the left. The right knee was partially flexed and the patient could extend it only at the cost of increasing the sciatic irritation. There was no visible or measurable atrophy of the right thigh or calf muscles. There was no palpable tenderness along the various spinous processes or interspinous ligaments. The lumbosacral joint was not sensitive to pressure. The right sacrospinalis muscle and right iliolumbar ligament were painful to fairly moderate pressure. The point of greatest tenderness was over the area of the right sacro-iliac joint. An effort to bend forward while keeping his knees extended was immediately inhibited by acute pain which he localized in the region of the right sacro-iliac joint. In the sitting position he could flex his spine more readily although the list to the left persisted. Straight leg raising was possible on the right to only about 20° when pain in the region of the right sacro-iliac joint was experienced. Straight leg raising on the left to 85° caused contralateral pain—that is, pain on the right side in the region of the sacro-iliac joint. The knee jerk and ankle jerk on the right side were normally active. Sensory tests for pain and temperature showed no untoward findings.

We reviewed his history. General health had been excellent and review by systems revealed no conditions which might have given rise to a symptomatic backache. There had been no previous attacks. The onset was associated with a definite act involving physical exertion, yet this was voluntary and not especially drastic or forcible. We had examined the patient. He presented the typical picture of a low back syndrome. All signs pointed toward abnormal sensitivity and palpable tenderness about the right sacro-iliac joint. There was sciatic irritation but neurological examination was negative. His x-ray plates included anteroposterior, lateral and oblique views of the third, fourth and fifth lumbar vertebrae and the first and second sacral segments. Well visualized were the lower lumbar vertebrae, the sacrum in its upper portion, the intervertebral spaces, the lumbosacral and sacro-iliac joints, and the lower lumbar interlaminar facets. The lumbar vertebrae were of normal outline without any lipping of their edges or other malformation; There was no narrowing of the intervertebral spaces; no congenital anomaly; the sacro-iliac joints appeared free of any gross bone pathology. In the lateral view the lumbosacral joint was not hyperextended; there was no spondylolisthesis; the intervertebral discs presented normal dimensions. In the oblique views no evidence of apophyseal subluxation was present. The x-rays then were negative for bone and apparently also for disc pathology.

Equipped with such informative data, it was reasonable to favor the tentative diagnosis of acute low back strain involving the ligaments and contiguous muscles of the right sacro-iliac joint accompanied by reflex irritation of the right sciatic nerve. The test for such a diagnosis lies in the response of the lesion to conservative therapy. The patient was manipulated without anesthesia with his full cooperation so far as voluntary or self induced relaxation is concerned. He obtained immediate respite from the acute pain. And as is characteristic with this type of lesion, he sensed a readjustment or realignment of structures which

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previously had given him the feeling of impingement. With the acute pain subsided and the muscle spasm lessened, he moved about more comfortably and with less apprehension. He was placed on the post-manipulative routine of rest during the period of immediate convalescence such rest being obtained on a solid bed (thick, tightly compressed felt mattress supported by fracture boards); infra red heat every four hours for thirty minutes to the low back, the latter being flexed over crisscross pillows with the patient in the prone position; massage to the paravertebral and gluteal muscles; and straight leg raising exercises performed alternately twice a day. When a patient has therefore been manipulated for an acute strain, the most important point in post-manipulative therapy is the patient's temporary cessation of activity.

GROUP II. *Conditions responding to manipulation with immediate partial relief and with progressive correction over a period of time.*

Into this group fall the more chronic cases of lumbosacral and sacro-iliac strain. These may represent instances in which the patient has neglected to seek medical aid at the onset of the condition or in which failure to give relief at the onset has occurred. The list which was produced by acute muscle spasm in the beginning persists now because of adaptive muscle contractures. Time is necessary in order to overcome the contractures and to reestablish muscle balance. Low back pain from postural lordosis fits into this same category. The contracted sacrospinalis muscles and very often the contracted tensor fasciae obviate any possibility of immediate or dramatic recovery. So, too, with the minor types of congenital anomalies. Sacralization of a transverse process may be a source of strain operative over an extended period of time. Then there are the cases of an arthritic type, slowly progressive, upon which has been superimposed acute or chronic strain such as occurs in various occupational activities. Roentgenograms of this latter type may disclose definite and often marked arthritic changes.

Illustration. A recent case coming under our supervision was that of a dentist some 52 years of age whose low back disability was becoming progressively more aggravated by the necessity to stand for prolonged intervals. The complaint had been present off and on for a period of years. At one time or another he had received most of the known forms of conservative treatment. An examination of his back failed to reveal any list, scoliosis or paravertebral spasm. Rather marked postural defects were evident,—a forward inclination of the head and neck, round back, flattened chest, marked anterior pelvic tilt with pendulous abdomen. The left shoulder was somewhat higher than the right,—this the patient described as an occupational deformity. His posterior superior spines were level as were also his gluteal folds. The area of greatest palpable tenderness was over the lumbosacral joint. There was also definite tenderness over his right sacro-iliac joint. Flexion of the spine in the standing position revealed marked lumbar rigidity accompanied by pain at the level of the lumbosacral joint. Sitting flexion yielded approximately the same effects. Hyperextension in the standing position evoked pain at the level of fifth lumbar and first sacral. Straight leg raising on the right was slightly less than on the opposite side and pain was felt in the region of the right sacro-iliac joint. The roentgenograms disclosed arthritic changes of a most marked degree involving the lumbosacral joint. Gentle manipulation without anesthesia appreciably mobilized his low back and afforded him some immediate comfort. (Incidentally, it is quite possible that manipulating a back of this pathology under anesthesia, thus eliminating the delicate check which the patient's voluntary actions supply, might precipitate an acute back condition rather than alleviate a chronic one.) Continued mobilization plus the other post-manipulative measure of therapy afforded him sufficent comfort to continue meeting the demands of his daily routine. A low back, well-stayed garment tailored to the patient by our corsetiere served to save him further episodes of pain. In these instances, the eventual need for a lumbosacral fusion is always in the foreground.

GROUP III. *Conditions responding to manipulative therapy with some immediate relief but no appreciable correction over an extended period of time.*

Into this group fall (1) cases of ruptured intervertebral discs or prolapsed nucleus pulposus and (2) the facet syndromes.

1. Ruptured intervertebral disc or prolapsed nucleus pulposus. As was pointed out in a recent communication, this type of intraspinal pathology produces a clinical picture quite similar to low back disability resulting from a number of other different causes. There is the recurrence of low back attacks of increasing severity, often an acute agonizing type of pain in the low back with sciatic irritation and the utter lack of response to all types of conservative treatment. We have gently manipulated a number of these cases without anesthesia and obtained a slight transient relief from the acute pain but another paroxysm might immediately ensue so as to make this effort for all practical purposes ineffectual. The definite diagnosis of these conditions can only be made by visualizing a defect in the intraspinal lipiodol or air shadow, such defect being seen at an intervertebral level in different views when the patient on a tilting table is being subjected to fluroscopic study. "Spot" roentgenograms are made at different angles to verify the fluroscopic findings. Other physical findings such as neurological changes and the total protein concentration in the spinal fluid are less constant and dependable as accurate diagnostic signs.

Illustration. A woman 35 years of age presented herself several months ago complaining of pain in the lower part of her back. Questioned about the duration of the complaint she stated she was entirely well until four years ago when she began experiencing an aching discomfort in the back at irregular intervals. She further noted these became evident during periods of fatigue. She received treatment intermittently from her physician for this "rheumatic" complaint. The symptoms persisted and last July (a year ago) she underwent an operation for removal of adhesions and complete hysterectomy. She returned home and in her first attempt to get about she was seized with pain in the lumbosacral region radiating down the right thigh which, while varying in intensity, has never completely disappeared. The onset of the pain necessitated her returning to bed where she remained through August until the latter part of September. She gradually assumed weight bearing and got about after a fashion until the following May when increasing severity of the pain required a period of bed rest for twelve days. The last attack occurred this past October confining her

to bed until January when her local physician referred her to us.

At the time of the initial examination, her standing position was characterized by a list toward the left. The paravertebral muscles were taut on both sides, somewhat more so on the left. The right shoulder was higher than the left. The posterior superior spines were equal. She apparently bore weight equally on both lower extremities both knees being held in extension. There was no visible or measurable atrophy of thigh or calf muscles. She referred the site of pain and greatest tenderness to the lumbosacral joint level. This pain radiated down her right thigh and leg. The pain seemed worse to her in the sitting position on a straight chair. In walking she noted a weakness of the right foot. In lying down, the most painful position was on her back, the most comfortable one being on her side with the thighs flexed. She complained of extreme sensitiveness of the skin of her right thigh, so much so that she could "scarcely bear" to fasten her hose supporters.

The resume of her back complaint and findings at the time may be summed up as follows: a low back pain situated at the lumbosacral level attended with right sciatic irritation; repeated attacks of increasing severity; onset unattended by trauma so far as patient can recall; localized tenderness on percussion over the fifth lumbar vertebra and first sacral segment; lumbar pain and rigidity on forward flexion; straight leg raising limited on the right side; positive Lasegue test on right side; diminished knee jerk and absent ankle jerk on the right side; paresthesia to the following extent: diminution in sensation (pin prick) over the posterior thigh corresponding to sacral 1 dermatone and some diminution in sensation over the heel and outer three digits of the right foot. Let us progress to the examination of her x-ray plates. The anteroposterior view suggests a diminution in the intervertebral space between the fourth and fifth lumbar vertebrae. If you will now glance at the lateral view you will agree that this impression is borne out very definitely. A lumbar puncture was done and the findings were indifferent: no evidence of block and a total protein concentration of 30 milligrams percent. (The total protein may be lowered as much as 20% when taken after the Queckenstedt is done. Fluid for total protein determination should therefore be withdrawn before making this test.)

Disregarding the spinal fluid findings because of the definite neurological findings in the physical examination of the patient, lipiodol to the amount of 5 cc. was introduced into the lower part of the spinal canal and fluoroscopic studies made on a tilting table. Studied from different views and with the patient in different positions, a constant unmistakable bilateral defect at the level of the intervertebral space between the fourth and fifth lumbar vertebrae was demonstrated. "Spot" roentgenograms were then made to verify fluoroscopic observations.

An interesting experiment was carried out during the fluoroscopic examination of this patient. After the defect had been amply seen and studied, the roentgenologist, Dr. Wendell Scott, of the Mallinckrodt Radiological Institute of Washington University Medical School, had the patient flex her spine. The defect was seen to disappear, the outline of the lipiodol becoming perfectly straight. Obviously the ruptured disc and nuclear material *had been reduced* by this manipulation of the spine. Then the patient was asked to hyperextend her spine and the definite defect in the lipiodol again became evident. This result was interpreted on the basis of the nuclear material *having been extruded again* into the canal by the maneuver of extending the spine from the flexed position.

At the time of the laminectomy the fragmented portion of the inter-vertebral disc was found to be extruded and this was removed.

2. The facet syndrome. Disturbances in the intervertebral facets result from an abnormal relationship between two adjacent vertebral bodies. A marked lumbar lordosis alters the normal alignment of the intervertebral joints and throws strain on their ligaments. Likewise, a thinning of the intervertebral disc, whether because of a ruptured nucleus pulposus or fibrotic changes in the disc itself, will produce an apophyseal subluxation. Such a subluxation may cause low back pain because of strain upon the joint capsules, by decreasing the size of the intervertebral foramina or because of impingement of the ends of the articular processes either against the pedicle above or the lamina below. Arthritic changes in these posterior spinal articulations may produce a similar clinical picture. In some instances gentle manipulation without anesthesia relieves the sharp pain of an acute attack. Although the exacerbations have been controlled by means of such conservative measures, we have in all instances acquainted the patients with the future possibility of a necessary fusion operation.

Illustration: A young neurosurgeon while leaning over the wash basin was seized with such severe pain in the right low back that he fell to the floor. The pain remained acute for several days and then subsided to a continuous ache. Three weeks after the onset, a second attack occurred but lasted only several hours. The present attack, six weeks after the initial attack, was of some 48 hours duration. The patient on physical examination pointed to the left sacrospinalis muscles at the level of the transverse process of the fourth lumbar vertebra and to the lumbosacral joint as the areas of pain. Definite tenderness to palpation was elicited at these points. In the standing position, a list to the right was evident and this was increased with flexion of the spine.

The roentgenograms disclosed chronic arthritis and obliteration of the apophyseal joints between the fourth and fifth lumbar vertebrae and the fifth lumbar vertebra and sacrum. He was manipulated several times with relief from the aching pain and muscle spasm. His list was almost completely effaced at the end of 48 hours. He was instructed in the proper manner of executing ordinary daily movements so as to avoid future attacks. He understood the vulnerable nature of his back lesion and the possibility for surgical intervention at a later date.

GROUP IV. *Conditions responding to manipulation with immediate relief and correction for a temporary interval but recurring attacks of increasing severity.*

This group comprises (1) sacro-iliac subluxations of chronic instability; (2) spondylolisthesis and spondyloschisis.

1. Sacro-iliac subluxations. Subluxations of the sacro-iliac joint of chronic recurrence, in contradistinction to acute sacro-iliac strains, are very

often of such pronounced instability that the patient is threatened with pain and disability whenever undue physical activity is attempted. The roentgenological technique of Chamberlin is an aid in demonstrating the presence of these lesions. According to this technique, sacro-iliac joint motion is demonstrated at the symphysis pubis. Toward this end, stereoscopic views of the pelvis (posterior projection) are studied for asymmetry, then a view of the patient's pelvis is taken (anterior projection) with weight bearing on one lower extremity, then on the other. By measuring the degree of movement of the symphysis pubis in these plates, the extent of sacro-iliac motion is determined.

2. Spondylolisthesis and spondyloschisis. Spondylolisthesis occurs when a separation or break occurs in the laminar arch thus "untying" the involved vertebra and allowing it to dislocate forward on the vertebra below. This break in the laminar arch or pedicle may be a congenital defect or an actual fracture resulting from trauma. It is not always easy to differentiate the two types. For in the former type, there might have been a fibrous defect which was ruptured by a traumatic incident and in the latter, the arch may have been normally intact until fractured by the traumatic incident. Either condition lends itself to conservative management by various methods ranging from sustained traction on the thighs with the hips in flexion to manipulation with or without anesthesia. In those types of congenital etiology, the effectiveness of conservative therapy is generally not permanently sustained and operative fusion must be done. In the cases of frank fracture, immobilization in plaster for six to 12 weeks following satisfactory reduction should suffice so far as corrective endeavor is concerned.

Illustration: A young girl 19 years of age dated the onset of her low back complaint to an incident two years before at which time she fell and injured her back. Since then attacks occurred every several months. She found that an attack could be precipitated by leaning forward and that seemingly, this could be reduced by lying down in bed. She described the pain as being in midline at the lumbosacral level. There was no radiation down the thigh but during an attack the pain radiated on either side anteriorly to the abdomen and groin. Roentgenograms disclosed a spondyloschisis and spondylolisthesis involving the fifth lumbar vertebra. Manipulation helped and tided patient over an attack but would not prevent recurrences. A spinal fusion was performed.

GROUP V. *Conditions in which manipulation is not only ineffective but harmful and therefore contra-indicated.*

These conditions include acute compression fractures of the vertebrae, acute arthritic processes, tuberculosis, luetic disorganization, advanced rarefaction, etc.

Summary

GROUP I. Conditions responding to manipulation with immediate relief and correction. Acute sacro-iliac and lumbosacral strains.

GROUP II. Conditions responding with immediate partial relief with progressive correction. Postural defects, contracted sacrospinalis muscles, contracted tensor fasciae, sacralization of transverse process, selected cases of arthritis, chronic lumbosacral and sacro-iliac strains.

GROUP III. Conditions responding with some immediate relief but no appreciable correction over an extended period of time. Ruptured intervertebral disc or prolapsed nucleus pulposus, and facet syndromes.

GROUP IV. Conditions responding with immediate relief and correction for temporary interval but recurring attacks of increasing severity. Sacro-iliac subluxations of chronic instability, spondylolisthesis and spondyloschisis.

GROUP V. Conditions in which manipulation is not only ineffective but harmful and therefore contra-indicated. Compression fractures of vertebrae, acute arthritic processes, tuberculosis, luetic disorganization, advanced rarefaction.

Cardiovascular Emergencies

—Recognition and Treatment—

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CARDIOVASCULAR emergencies constitute one of the most important phases of medical practice and continue to become more important as the years go by. These emergencies are of varied nature and must be properly interpreted before it is possible to institute intelligent treatment.

First of all, it might be well to classify these emergencies under certain headings for the purpose of discussion.

First, there are those which are the results of structual disease of the heart; second, those arising from disturbances of cardiac rhythm; third, those of vascular origin; fourth, those of nervous of reflex origin; and fifth, those, the result of drug intoxication or idiosyncracy.

Under the first classification of structural disease of the heart we find those emergencies of pericardial, myocardial, and endocardial origin.

Occasionally pericardial effusion accumulates rapidly enough to endanger the life of the patient. The diagnosis of pericardial effusion in most instances is not too difficult, particularly, if there is the pathognomonic to and fro superficial, scratchy, friction rub, heard best near the left border of the sternum. Associated with this we find an enlarged cardiac silhouette of pyramidal or water-bottle contour with diminished or absent pulsations on fluoroscopy. The heart tones become faint, and a paradoxical small pulse develops. Orthopnea, cyanosis, tachycardia, distention of neck veins, and fall in blood pressure is noted. When the fluid in the pericardium accumulates to a point where there are signs of cardiac tamponage, parecentesis should be done immediately.

We come now to the cardiovascular emergencies of myocardial origin with which we are all very familiar.

Read at the Fifty-First Annual Meeting of the AMERICAN ASSOCIATION OF RAILWAY SURGEONS, Chicago, September 18, 1940.

I am speaking now of congestive heart failure with involvement of the pulmonary circulation, or left sided heart failure. The recognition of this state of affairs is quite well known and is manfested by dyspnea on exertion, orthopnea, cardiac asthma, Cheyne-Stokes' respiration, cough, rales at the lung bases, diminished vital capacity, and increased vascular shadows in the lungs. At times the addition of such burdens as fatigue, tachycardia, infection and coronary thrombosis to a diseased but compensated heart may precipitate acute congestive failure. Then, all too frequently, we encounter the patient who is having an attack of acute pulmonary edema with the usual signs of prostration, marked dyspnea, cough with abundant, pink, frothy sputum and cyanosis. The lungs are filled with fine and coarse rales. The blood pressure is often elevated during the attack.

The treatment of congestive failure demands absolute rest, digitalis, and diuretics. Rest is obtained in a quiet room, in a comfortable bed with back rest, and by sedatives. Morphine, grs. 1/6 to ¼, is used freely and judiciously the first two or three days and then supplanted by phenobarbital or sodium bromid, or both. Digitalis still remains the most important drug in the treatment of congestive heart failure and prompt digitalization is indicated. This is accomplished by giving about 2 gms. of digitalis in pill form over a period of three or four days, using one-fourth the total dose the first and second days. On the third day it may be necessary to reduce the dose, this being judged, however, by the condition of the patient or the rate of the heart at the apex. Occasionally it becomes necessary to give digitalis by rectum. The dosage is just the same as that used by mouth, except that the tincture is diluted in water and injected.

When the situation is very urgent, the intramuscular and intravenous routes should be used. The usual initial dose is 0.5 gms. to 1 gm. An effect may be expected in one to two hours when it is injected into the buttocks and within 15 to 30 minutes when given intravenously.

Improvement in the patient after this initial parenteral dose often enables one to complete the digitalization by the oral route.

The use of diuretics in the treatment of congestive failure is next in importance to digitalis and are best used after digitalization has been completed. Those most often used are theobromine, sodium salcylate or (diuretin) 1 or 2 gms. three times a day and theophyllin (theocin) 0.2 or 0.3 gms. T.I.D., for one or two days a week.

If results from these drugs are not too effective, we may use one of the mercurial diuretics such as salyrgan or mercupurin. They are given in doses of 1.0 to 2.0 cc. either intramuscularly or intravenously. Suppositories (mercurin) are now available and often give the desired diuresis. The preliminary administration of ammonium chlorid, 1 gm. Q.I.D., for four days may even improve the diruetic effect of the mercurial drugs and is worthy of trial.

When acute pulmonary edema is encountered, the treatment is directed toward diminishing the flow of the blood into the engorged pulmonary circuit. This may be accomplished by the use of nitroglycerine, 1/100 gr., or by the application of tourniquets to the four extremities. Occasionally a phlebotomy of 300 to 500 cc. is extremely helpful. Oxygen is beneficial when cyanosis is present. This is one of the conditions in which the intravenous administration of digitalis is indicated in the dosage previously outlined. Because of their prompt and powerful action, either pure crystalline ouabain or strophanthin intravenously in a dose of 0.5 mgm. may be necessary and may be repeated in 12 hours provided digitalis has not been used.

It is surprising to find that the prompt administration of morphine and atropine sulphate is all that is required to relieve most of the less severe attacks of acute pulmonary edema.

The cardiovascular emergencies of endocardial origin are most often the result of aortic stenosis, mitral stenosis and subacute bacterial endocarditis. Aortic stenosis is of interest because of its ability to produce attacks of syncope and angina pectoris. It is recognized clinically by the presence of a systolic thrill and murmur in the aortic area, a faint or absent aortic second sound, enlargement of the left ventricule, low systolic and small pulse pressure, and calcification of the aortic valves on x-ray examination.

THE treatment is directed toward reduction of the patient's physical activity, thereby preventing attacks of syncope and anginal pain.

Mitral stenosis is of interest in this discussion principally because of a tendency for the formation of mural thrombi within the cavities of the auricles. From these thrombi, emboli may be dislodged into the pulmonary or systemic circulations with resultant pulmonary infarction or arterial occlusion. The same may happen as the result of emboli arising from valvular vegetations in subacute bacterial endocarditis. The clinical manifestation resulting from emboli obviously vary with the structure involved and will be discussed with their treatment, later on.

The diagnosis of mitral stenosis and subacute bacterial endocarditis in most instances is rather easy and needs no further discussion.

We come now to the cardiovascular emergencies arising from disturbances of cardiac rhythm. Of course, an emergency rarely exists with these disturbances unless there is underlying heart disease as evidenced by a history of substernal distress, cardiac enlargement, or the presence of a diastolic murmur. The recognition and proper treatment of these disturbances of rhythm will, in most instances, prevent acute congestive heart failure and prolong life.

Of these conditions, the development of sinoauricular tachycardia is the least important. However, in the rare case where there is evidence of an associated heart disease with limited coronary or myocardial reserve, acute heart failure may be precipitated and will need emergency treatment.

The diagnosis of sino-auricular tachycardia is generally obvious when we have a rapid, regular cardiac rate of 100 or over which varies with forced respiration. No treatment is necessary except with the development of acute heart failure.

The most common type of rapid heart action that is encountered is auricular paroxysmal tachycardia. It is characterized by the sudden onset of a very rapid, regular, cardiac rate of 160 to 220 which is not affected by rest or exercise. It may last from a few seconds to several days, terminating either abruptly or gradually.

The diagnosis is usually made with ease and treatment is generally rather simple. The most satisfactory treatment of this condition is stimulation of the vagus nerve. This may be accomplished by firm pressure, for a few seconds, over the carotid sinuses, by pressure on the eyeballs, or by touching the back of the throat so as to cause gagging or even vomiting. If the paroxysm is persistent and prolonged, more intense vagal stimulation is indicated. This is done by giving syrup of ipecac, 2 to 4 drachms, every two to four hours, or until vomiting ensues; or one may use mecholyl, 10 to 50 mgm., subcutaneously. The latter drug may cause sweating, nausea, vomiting, and sometimes collapse; but atropine sulphate, grs. 1/60, will control these signs. If there is no relief from these methods, quinindine sulphate, grs. 3 to 6, every two hours for three to five doses should be given. In the emergency quinine dihydrochloride, 7½ grs. may be given intramuscularly, but when the patient is in congestive failure, it is probably better to use digitalis.

The second most important but relatively uncommon rapid, regular rhythm encountered, is auricular flutter. The majority of patients who show aruicular flutter have heart disease and it is evident that any prolonged cardiac strain is to be avoided. The clinical recognition of auricular flutter is quite difficult. Occasionally, however, one may suspect that such a condition exists, if on examination of the neck, one sees several venous pulsations to each ventricular wave and also if pressure on the carotid sinus causes temporary halving of the heart rate. However, the diagnosis of auricular flutter is earily made by the electrocardiogram.

Digitalis still remains the drug of choice in the treatment of auricular flutter. The patient should be digitalized rather rapidly and digitalization should be maintained until the attack is over with the re-establishment of a normal rhythm. If the condition of the patient remains serious because of myocardial weakness, or if auricular fibrillation persists, digitalization should be maintained.

Occurring less frequently than auricular paroxysmal tachycardia and auricular flutter, but usually associated with even more serious disease of the heart, is another condition of rapid, regular, cardiac rate known as ventricular paroxysmal tachycardia. This condition is most often seen following recent coronary thrombosis or digitalis poisoning of a high degree. The occurrence of runs of ventricular extrasystoles in twos and threes is a warning that ventricular paroxysmal tachycardia is impending. The diagnosis can be made only with certainty by the electro-cardiogram. In the treatment of this condition we find that quinidine sulphate is quite effective. It is given in 3- to 6-grain doses at intervals of two hours for three to five doses and then according to the patient's needs and susceptibility.

The patient with marked slowing of the heart rate, the result of auriculo-ventricular heart block, often shows a tendency to syncopal attacks with or without convulsions. The condition then presents itself as a cardiac emergency and demands immediate treatment.

The clinical recognition of complete heart block is ordinarily a simple matter when the cardiac rate ognomonic accentuation or reduplication of the is 40 or less, and one finds on auscultation the path-first heart sound at the apex. In the emergency, the subcutaneous injection of 0.5 to 1 cc. of a 1:1000 adrenalin solution is often all that is needed. Occasionally ventricular standstill occurs during anesthetics or surgery and here again the injection of adrenalin directly into the ventricular cavity or ventricular muscle may be a life-saving measure. When cardiac standstill occurs during an abdominal operation, massage of the heart through the diaphragm may be of value. The restriction of the patient's activity and use of ephedrine sulphate, ⅜ to ¾ gr. T.I.D., by mouth often will be effective in preventing the mild attacks of syncope, the result of auriculo-ventricular block.

The onset of a rapid, irregular cardiac rate may impose an excessive load on a heart that is already damaged and lead to rapid failure. Auricular fibrillation occurring under the above circumstances needs early recognition and treatment. The diagnosis is usually rather easy, especially if one remembers that any grossly irregular cardiac rate of 120 or over is most certainly the result of this condition. Here again the diagnosis is made certain by the use of the electrocardiogram. The treatment of preference is ordinarily digitalization, especially so if there is underlying evidence of heart disease or the existence of an emergency cardiac state. There is no place for use of quinidine under the above circumstances.

FINALLY, let us consider a rare but serious cardiac irregularity of ventricular origin; namely, ventricular fibrillation. It is very unusual to find this condition in clinical practice because, in most cases, it is the cause of instant death. However it is most often seen in coronary artery disease and electrocution. Occasionally recovery takes place, and then quinidine sulphate may be of some value in preventing further attacks of ventricular fibrillation.

The emergencies of vascular origin constitute one of the commonest and most important groups seen in the practice of medicine. They include such conditions as peripheral vascular collapse or shock, coronary and cerebral thrombosis, cerebral hemorrhage, spontaneous subarachnoid hemorrhage, dissecting aneursym, and emboli.

First let us consider peripheral vascular collapse or shock, a condition well-known to us all, but which is far too often unrecognized and poorly treated. The characteristic signs are pinched features, pallor, prostration, sweating, cold extremities, rapid thready pulse, poor heart tones, and low blood pressure. The treatment, as Atchley has well discussed, is toward overcoming the disparity between the volume of circulating blood and the capacity of the vascular bed that contains it. The best method of increasing blood volume is blood transfusion, but isotonic salt solution and hypertonic sugar solution are often very effective and more quickly available.

In shock the head of the patient is lowered and the body is kept warm. Stimulants such as caffeine, sodii-benzoate, strychnine, and camphor are often of value. For undue restlessness or irritability morphine sulphate may be necessary and helpful.

The second group of vascular emergencies include such frequent conditions as coronary and cerebral thrombosis.

The recognition of acute coronary thrombosis in its typical form is familiar to most everyone. The clinical picture of severe substernal pain occurring often at rest and associated with signs of shock will direct one's attention to the heart. The electrocardiogram is an important aid in the diagnosis and location of the area of infarction. The emergency measures used here are the same as those used in acute congestive heart failure, except that digitalis is withheld until signs of failure appear. Morphine sulphate is used freely and oxygen is of value when there is cyanosis. Aminophyllin may be of some value but it has not been generally accepted as a necessary drug in the treatment of acute coronary thrombosis. When multiple extrasystoles follow coronary thrombosis, it is probably wise to give quinidine sulphate, grs. 3 T.I.D., to prevent the onset of ventricular tachycardia or ventricular fibrillation. Some authors recommend the prophylactic use of quinidine sulphate in all cases of acute coronary thrombosis.

It is often difficult to differentiate clinically between cerebral thrombosis, cerebral hemorrhage, and cerebral embolism. However, cerebral thrombosis is most common in people past middle life and is usually the result of arteriosclerosis, either with or without hypertension. The onset here is usually gradual with paresthesia of the extremities, vertigo, and thick speech, preceeding the onset of paralysis.

In the third group we find those emergencies arising from complete or partial rupture of a diseased blood vessel. Of these, the cerebral hemorrhage occurs most frequently. The onset of cerebral hemorrhage is more rapid and the coma deeper than in the patient with cerebral thrombosis. Here the breathing is stertorous, the pulse slow, the face flushed, the pupils are often unequal, and the corneal reflexes are absent. The patient is shocked and in this state, the control of the sphincters is lost. The hemiplegia develops rather rapidly.

The treatment of acute attack of cerebral thrombosis or cerebral hemorrhage calls first of all for bed rest and sleep. The application of an ice bag to the head, sedatives for restlessness, cleansing enemas, and frequent change of position are important considerations in the treatment. If there is difficulty in swallowing, fluids are given either by nasal catheter or bowel. If the blood pressure is high and hemorrhage is suspected, venesection of 300 to 500 cc. of blood may be quite valuable. Relief of cerebral edema or increased cerebral pressure may be obtained by repeated spinal punctures and intravenous injections of magnesium sulphate 20 cc. of 10% solution B.D. or T.I.D. After three weeks the patient is allowed out of bed and massage and passive motion started on the paralyzed extremities.

Somewhat infrequently we encounter the patient with spontaneous subarachnoid hemorrhage. The majority of these cases are found in young persons. The onset is usually sudden with dizziness, headache, drowsiness or stupor, neck rigidity, ptosis of the lids, moderate fever, and slow pulse. The spinal fluid is invariably bloody. The treatment consists of bed rest, limitation of fluids, repeated spinal punctures, and magnesium sulphate by mouth or vein.

IF ONE should encounter a case which simulates acute coronary occlusion, but which lacks the characteristic electrocardiographic findings, careful consideration should be given to dissecting aneursym of the aorta — a rather rare but serious vascular disease of this group. The onset is sudden with severe agonizing pain which varies with the site of the aneursym and which exhibits a migratory tendency often radiating to the back, flanks, or leg. There is also pallor, prostration, and often loss of consciousness. The blood pressure is elevated and remains so even though prostration is extreme. The treatment is directed toward the relief of pain by opiates and the reduction of the blood pressure by vasodilators (nitrites).

The fourth group of emergencies, the result of embolic occlusion, includes some of more serious vascular accidents. Of these, pulmonary embolism is the most important and alarming. It is most commonly seen following surgical operations, pregnancy, thrombo-phlebitis, and as a complication of rheumatic heart disease, especially mitral stenosis with auricular fibrillation. It is usually characterized by the sudden onset of chest pain, extreme breathlessness, and cough. The familiar signs of shock are often seen and cyanosis is variable. When infarction of the lung results, expectoration of bloody sputum is not uncommon, but it is not an early sign.

The clinical picture may, at times, closely resemble that of coronary occlusion, but careful analysis of the situation and the finding of a focus from which an embolus may have arisen enables one to arrive at a correct diagnosis. The treatment, of course, is necessarily urgent and energetic. The immediate relief of pain by morphine sulphate and the treatment of the associated

shock is paramount. The patient is kept absolutely quiet and warm, the head low, and oxygen inhalation is continued if there is an associated dyspnea and cyanosis. If signs of right heart failure occur, one may try digitalis, 7½ grs., intramuscularly twice in four hours. If the patient survives the acute state, further treatment is directed toward keeping the patient quiet in order to prevent, as much as possible, the dislodgment of other emboli.

Cerebral embolism is more likely to be seen in the young individual who has valvular heart disease, especially mitral stenosis and subacute bacterial endocarditis. In this condition the onset of hemiplegia with or without unconsciousness is quite sudden. The presence of heart disease and other embolic evidences, the result of bacterial endocarditis, aid in the diagnosis. The treatment, of course, is directed toward the primary disease and the cerebral manifestations as described previously.

The development of sudden circulatory emergencies, the result of embolic occlusion in a peripheral vessel, again are almost invariably due to organic heart disease. These emboli may involve the limbs, intestine, kidney, or spleen, and the clinical signs vary with the structures involved. The sudden occlusion of a vessel to an extremity produces severe agonizing pain, pallor, coldness, and absence of arterial pulsation in the affected limb. The treatment consists of relief of pain, heat to lower half of patient's body and leg, lowering of the limb, and the use of papverine hydrochloride, gr. ¼, dissolved in salt solution, intravenously. The Pavex machine may be of value in either case. During the first few hours embolectomy offers a good chance of recovery in selected cases. If gangrene develops, surgical treatment is imperative.

The occlusion of a mesenteric vessel is of serious import and requires accurate diagnosis. It is characterized by the development of sudden onset of severe pain in the abdomen, marked general or localized abdominal tenderness, and rigidity, and later on, signs of intestinal obstruction. The immediate treatment is symptomatic with morphine, but the earlier operation is performed the greater the chance of recovery. Mesenteric thrombosis occurring as a complication of subacute bacterial endocarditis is most unfortunate and one is hardly justified in recommending surgery.

The fifth classification includes such cardiovascular emergencies that are of nervous or reflex origin. These include benign and carotid sinus syncope. The recognition and treatment of benign syncope needs no further discussion.

In recent years our attention has been called to a condition known as carotid sinus syncope. It apparently is the result of irritation of the nerves surrounding the carotid sinus. When syncope can be ascribed to sensitivity of this sinus the administration of ephedrine sulphate, grs. ⅜ to ¾ T.I.D., may be effective in preventing these attacks. In the severe case it may be necssary to resect the nerve plexus from the carotid artery.

In closing I would just like to mention that occasionally one sees cardiovascular emergencies arising from the use of two well-known cardiac drugs—digitalis and quinidine. The development of a coupled rhythm, auriculo-ventricular block, paroxysms of ventricular tachycardia, and cardiac standstill in a patient under treatment with either of these drugs calls for their prompt withdrawal. This is followed by a cessation of the signs and symptoms and recovery of the patient.

The Industrial Hygiene Engineer

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THE huge suspension bridge constructed by experienced engineers over Puget Sound performed its function as an essential link in a highway important to national defense. But when it developed symptoms suggestive of a functional disability, there was an immediate need for personnel trained in the control of undulations in suspension bridges. The lack of such trained personnel capable of correcting the potential collapse hazard permitted this finely fabricated structure to plunge its road-bed into the sea.

It was not so many years ago that lack of trained industrial hygiene personnel permitted the collapse of certain industrial concerns, seriously impeded the introduction of marked economies affecting, in one well-known instance, every automobile operator in the country, and contributed to the existence of environmental factors involved in the diminution of the average life-span of the industrial wage earner by several years.

Let me first indicate something of what has happened where there has been lack of trained industrial hygiene engineers. The present situation of supply and demand of such trained personnel will then be discussed, followed by a concept of needs for the immediate future based upon the observations of a number of persons in the country who are close to present, fast moving developments. Methods of meeting these needs will necessarily be touched on somewhat sketchily since specific plans are now in state of development.

Lack of Trained Industrial Hygienists

A NUMBER of the more important of the occupational disease situations have the characteristic of lying dormant so far as exciting attention is concerned, and then, due to a combination of circumstances, both physiological and economic, they spring into painful prominence.

The first of such situations in the personal experience of the writer occurred some fifteen years ago. Artificial leather manufacturers were advised by experienced engineers that uniform drying of

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the dope applied to the fabric base could best be obtained with a slight positive air pressure in the drier. They were advised by experienced chemists that benzol was the optimum diluent to use in the make-up of the dope. The trained industrial hygiene engineer had not entered the picture. What happened?

At first, nothing "happened." But later investigation showed that labor turnover was high. Workers came to realize that they did not feel as fit after working around the coating machines for a while, and they left for other jobs. Then a man died, and another, and another. Things were now beginning to happen!

Management called in ventilation engineers. The state labor department held a meeting of the plant executives and told them to find some other solvent or shut up shop. The insurance companies boosted their compensation rates to help meet the high losses which they were paying. Now, every man whose health was being affected collected compensation, as was his just due. And many men who suffered pains and aches not caused by the occupational exposure also submitted claims for compensation. But the situation began to get out of hand when a worker who had been fired for stealing a bolt of fabric presented a compensation claim the next day and settled for $2700.00 before the case had been thoroughly investigated. As the claimant asserted, benzol was still in use at this plant, but he did not add that its only use was as a denaturant of the alcohol—to the extent of 2%.

It is my contention that this is the type of situation which can occur when there is no trained industrial hygiene engineer to maintain constant supervision over industrial operations. Just such a situation has happened on a number of occasions, and it will probably happen again.

How much more intelligent is the control of the occupational disease exposure where the trained industrial hygienist makes a survey of operations before the occupational disease occurs. In one artificial leather plant not long after the situation related above, ventilation was applied according to best engineering practices but benzol vapor determinations showed exposures to be excessive. Adjustments were made in exhaust ventilation to provide negative air pressures inside the driers, and in accordance with most economic production, benzol was continued as the solvent after atmospheric determinations showed vapor concentrations to be well within safe limits. This was before the days of the urine sulphate test but were the beginnings of the modern technique now being utilized by industrial hygienists all over the country.

We are all familiar with the silicosis situation which came to light through proper diagnosis by trained physicians and then, with the economic stress of the depression, assumed racket proportions, and actually put some concerns out of business.

With trained industrial hygiene engineers on the job, backed up by physicians trained in correct correlation of roentgenogram with actual pathology, the possibility of another silicosis situation of such magnitude is not great. But there is the danger that excessive dust exposures will be tolerated in these times of production pressure without calling an industrial hygiene engineer into the picture, or through deferring action on his recommendations. Men will continue at work with gradual development of silicosis. Comparatively few, it is hoped, will incur tuberculosis or other infection necessary to precipitate disability during this period.

But when employment falls off, when the pinch of decreased wages causes reduction in healthful living standards, what will happen? It is well known that more crowded living conditions, poorer nutrition and inadequate clothing lead to increased incidence of tuberculosis among general populations, and those men having silicosis will have a greatly increased chance of tuberculosis being superimposed on the silicotic condition with resultant disability or death.

Now is the time to assure that the great number of exposures to potentially injurious materials, processes and conditions incident to economic productions are kept under control so that the health of our workers may not be impaired — and the health of our workers is a vital element not only in national defense at present but it is equally vital to the security of our entire population in years to come.

Trained Industrial Hygiene Engineers

WITH no further demonstration of what actually has happened where modern industrial operations have been conducted without benefit of the trained industrial hygienist and with the simple statement that stepped-up production is introducing greatly increased possibilities of injury to health, let us examine present resources in trained industrial hygiene engineers.

As recently as the middle of the last decade only three states included industrial hygiene engineers in their health or labor departments. At that time the value of these services met wider recognition and now there are 39 official industrial hygiene units located in 29 states and two territories. All of these units include industrial hygiene engineers, the number of them depending more upon the extent of development of the service in a given area than upon the actual need for the work. In no case, however, has any industrial hygiene unit been over-manned and in every case the unit has had to watch its program carefully to avoid being taken up entirely with occupational disease exposures requiring urgent attention but which might not be of as fundamental importance as other activities which were broader in their scope.

Only the largest industrial concerns have full time industrial hygiene engineers and chemists with laboratory and field equipment. One large corporation includes some two dozen such engineers in its organization. In many more concerns, the accident prevention department is charged with control of occupational disease hazards. In still others, the engineering department has the re-

sponsibility to install and maintain operations so that the exposures to potentially injurious materials will be minimized. Where potentially harmful gases or vapors are evolved in processes such as in viscose rayon manufacturing, the chemical laboratory may make routine checks of extent of exposures and report their findings regularly to management.

The system followed by some concerns where there are several separate departments or plants is to place an industrial hygiene service on call when, as, and if the superintendant of that unit makes a formal request. This system has the advantage of encouraging the superintendent's cooperative interest in whatever preventive suggestions may be made as a result of the industrial hygienist's survey, but it has the disadvantage of insufficient contact with potential hazards which can be overcome by a more direct system with retention of the superintendent's cooperative spirit through demonstrated practical work on the part of the industrial hygienist.

A preferable method of procedure, which of course must be sufficiently flexible to meet individual needs, is one which is at present proving its value in industry. The industrial hygiene engineer is, to be sure, available to any of the plant superintendents on call. But his work does not begin and end there. He is kept continually informed by the purchasing department on potentially injurious materials coming into the plants. He knows where those materials go and what becomes of them. He has a record of potentially harmful processes being conducted with a schedule to check these at proper intervals.

He makes general surveys at regular periods since industrial operations are always in a state of change.

If an occupational disease case occurs — and it is the industrial hygienist's responsibility to see that it doesn't — facts are available concerning the man's exposure both as to concentration and duration.

These facts assist the physician in arriving at the proper diagnosis and treatment. Of greater moment, they permit setting up conditions to prevent occurrence of further injury to health from this operation or similar operations at other locations.

The vital role played by the physician is being omitted from this discussion as the next speaker is to tell of the need and availability of the trained industrial medical men, but we wish it to be clearly understood that the writer by no means underestimates the essential part of the physician in the industrial health picture.

A number of the compensation and group life and disability insurance companies provide the services of industrial hygienists to their policy holders.

McConnell[1] covered the services of these and other independent agencies in a comprehensive paper about two years ago. In addition to technical services backed up by laboratory and field determinations of potentially injurious materials, a large number of the insurance companies have provided their safety engineers with training in occupational disease hazards and the control of them.

Training Facilities for Industrial Hygienists

A SMALL number of universities are now providing training courses for industrial hygienists.

Most notable of these is the one at Harvard School of Public Health under the direction of Philip Drinker, Professor of Industrial Hygiene, which has been supplying well trained men to the field for many years. Others are the University of Pennsylvania Medical School, Department of Public Health and Hygiene, where industrial hygiene engineering is under the direction of Professor Theodore Hatch. Professor Donald E. Cummings, Director of the Division of Industrial Hygiene, University of Colorado Medical School, is amply qualified through his practical accomplishments in this field for training industrial hygiene engineers. The University of Pittsburgh Medical School and Johns Hopkins University have set up curricula, the latter largely for physicians in public health administration courses. Other institutions, the University of Michigan, for example, have also been interesting themselves in this field.

The Division of Industrial Hygiene of the National Institute of Health has presented brief courses in industrial hygiene over the last five years and has directed in-service training in many of the state units. The National Conference of Governmental Industrial Hygienists, sponsored by the United States Public Health Service, has through its annual meetings been doing much in furthering the training of industrial hygiene engineers attached to federal, state and municipal departments.

The American Industrial Hygiene Association was organized a year and a half ago to include industrial hygienists and others interested or active in this field. One of its objects is stimulating the dissemination of information among industrial hygienists through encouraging further attention to such publicatinos as the *Journal of Industrial Hygiene and Toxicology*, and INDUSTRIAL MEDICINE, particularly the quarterly Industrial Hygiene Sections of the latter periodical, through local section meetings of the American Industrial Hygiene Association in the more important industrial centers, and through its annual meeting which is assuming the nature of a post graduate institute of industrial hygiene.

Needs for Immediate Future

FIVE years ago, Gray,[2] in discussing the importance of industrial hygiene on the basis of administration of the Connecticut Bureau, pointed out that the prevention of occupational diseases necessitates technical personnel specially trained in the application of precise physical and chemical procedures to conditions affecting health in industrial environment.

One year ago, Bloomfield[3] stated that one of the primary needs today is a sufficient number of trained personnel, that courses for teaching the methods employed in the engineering control of health hazards in industry should be organized by our educational institutions as quickly as possible.

Nine months ago, Parran[4] asserted that we have a serious situation in this country in the inadequate number of persons qualified to work in industrial hygiene, that the facilities for training persons in this field are entirely too limited, and that additional universities should develop their facilities for teaching and training people in industrial hygiene.

And only this month, it was brought to the writer's attention that on the basis of an immediate need for 200 to 500 non-medical industrial hygienists, plans are now in progress for development of short, concentrated courses on the practical application of industrial hygiene at appropriate universities throughout the country.

These new recruits among the industrial hygiene engineers will be required primarily by the accelerating governmental program for national defense, by industry itself as it becomes involved in the introduction of new and expanded operations, by the official state industrial hygiene bureaus, and by the unofficial agencies. Of the governmental requirements, the Division of Industrial Hygiene of the National Institute of Health has submitted a program for the consideration of Congress which envisages the employment of some 15 additional engineers. It is the plan of this Division to provide training for these men within its own organization. The state industrial hygiene units have been canvassed and their needs call for 70 engineers and 34 chemists. The army arsenals and the navy yards, according to an authoritative estimate, can very well utilize approximately 20 engineers.

Production schedules need not and should not be maintained at the expense of the health of the worker.

During the last world war Hamilton[5] reported some three hundred cases of trinitrotoluene poisoning.

There need be no duplication of such experience either with this or other material with adequate trained industrial health personnel.

We should also consider the permanent needs for industrial hygiene engineers after the present emergency has passed. With stable industrial conditions, it is anticipated that there may be a tendency in two directions. The first, that many men with industrial hygiene training will tend to include other interests in their contact with industrial organizations. Some will become active in accident prevention in addition to the occupational disease prevention; others will become absorbed by personnel relations aspects of industrial health; still others will become engaged in plant control in its several phases.

There will still be an expanding need for the full time industrial hygienist as mechanization becomes more extensive, as present chemical materials are more widely used and new ones introduced, and as interest in maintaining high health levels increases.

The second tendency, which should be encouraged, is the inclusion in engineering curricula of instruction in sanitary air analysis and in industrial ventilation. Whether undergraduate engineers should also receive something in physiology or toxicology is debatable, but certainly chemists and chemical engineers should know the toxicological properties of the materials concerning which they have for the most part been instructed only in the physical and strictly chemical properties.

IN conclusion, it may be stated with certainty that there is not at present a sufficient number of trained industrial hygiene engineers to cover adequately the present exposures to occupational disease hazards. That this is a matter of no little moment is attested by the estimate of the National Institute of Health on the basis of actual surveys by state bureaus that one and a half million persons are exposed to hazards of carbon monoxide, three-fourths of a million to lead and its compounds, over one million persons to silica dust—to enumerate only a few of the most important.

Although present training facilities are recognized as inadequate to take care of the greatly increased need in the present emergency for industrial hygiene engineers, estimated at from 200 to 500 men, plans are now in progress for setting up short-time training courses in a number of universities.

In order to attract high-caliber men to the field of industrial hygiene engineering, caution should be observed not to flood the market beyond its saturation point. The demand for the full time industrial hygienist should be stimulated as actual need dictates. His services should not be limited to being on call, but he should be charged with the responsibility of controlling exposures which may lead either to a case of occupational disease or to lowered health level.

The trained industrial hygienist may in some instances find himself, either by inclination or as his individual situation develops, becoming interested in additional fields of safety engineering, personnel relations, or process control. And the engineer, both mechanical and chemical, should receive somewhere in their admittedly already crowded courses some instruction in the elements of industrial hygiene.

Every effort should be made to assure that capable men are given practical instruction and that plans for expansion fit as exactly as possible the actual needs of the national defense program.

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Registration of Physicians

—Under the Workmen's Compensation Law and Requirements for Advancement in Ratings and Qualifications—

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FROM time to time the Industrial Council has requested information concerning the standards set up by the State Medical Society and the various County Medical Societies on which the qualifications of specialists are based.

Dr. David J. Kaliski, Director of the Workmen's Compensation Committee of the Medical Society of the State of New York has very kindly furnished me with a set of standards and requirements which cover most of the major specialties and I quote them as follows:

Designation X: Under the designation X, we have included all physicians who do not confine their practice to a specialty, or to one particular field.

Designation S: Under the designation S, we have included all physicians who confine their practice to a specialty, or who limit their practice to one or more closely related fields of medical endeavor.

Requirements for Rating in Major Surgery

LICENSE to practice medicine in the State of New York.
2. Evidence of a surgical internship in an approved voluntary or municipal hospital. The length of intern service should extend over a sufficient period of time (from one and a half to three years) to enable the candidate to obtain adequate training in major surgical procedures under proper surgical auspices. This should preferably be followed by a surgical residency of one to two years, also in an accredited hospital, and under proper surgical tutelage.

Where the surgical internship is not regarded as adequate, consideration may be given to subsequent service in an accredited hospital as a member of the attending surgical staff, performing major surgical procedures independently.

Where the internship and residency period extend over a period of at least three to four years, and where during the residency period the candidate can present proof of having performed major surgical procedures independently and without supervision, a subsequent period of private practice of from one to three years will be required, depending on the experience gained during the training period and the submission of records of major surgical procedures performed without supervision subsequent to the training period.

Where the candidate has had only a single surgical internship of from one to three years, performing major operations under supervision, he shall give evidence of at least three to five years' experience in private practice as an independent operator with records of cases operated on and results.

In the latter instance, the physician shall present additional evidence in the shape of credentials from the attending surgeons on the staff of hospitals with which he was associated as to his surgical experience and results.

Candidates for the "XA" rating should present the above evidence of qualifications in surgery except that it is not necessary for the applicant to have had a seven-year interval after graduation from medical school. A minimum of five years after graduation should be required.

For the "SA" rating the candidate must present evidence of thorough preparation and qualification in surgery and shall devote his practice exclusively to surgery. He shall have had at least seven years' surgical experience, both in hospital and private practice subsequent to graduation from medical school; and should have an appointment in an approved hospital on the regular surgical attending staff enabling him to do independent major surgery and to keep abreast of surgical progress.

Candidates for both the "SA" and "XA" qualifications shall present evidence of training and experience in traumatic surgery during their training period and subsequent career in private practice.

Membership in the American College of Surgeons (or other accredited surgical societies membership which is predicated upon surgical qualifications or outstanding ability in surgery) may be accepted as evidence of surgical qualifications.

Requirements for Rating in Orthopedic Surgery

LICENSE to practice medicine in the State of New York.
2. Physicians who have had a regular orthopedic internship in an approved general hospital or orthopedic hospital for one or more years, and in addition three years' service on a regularly constituted orthopedic staff of a hospital accredited by the A.M.A. or the American College of Surgeons, and who shall have been in private practice, 50% of which at least consists of orthopedic surgery for a period of at least two years.

Physicians who have had an internship of one year or more in an accredited general hospital, and who in addition have been on the orthopedic service of an accredited hospital for a period of five years or more, and whose private practice consists of 50% or more orthopedic surgery. They shall have had at least two years of private practice in the United States.

Physicians who have received a surgical rating "A" and who are members of a duly constituted orthopedic staff of a hospital for a period of five years or more and whose private practice consists of 50% or more of orthopedic surgery.

Membership in the American Orthopedic Association, American College of Orthopedic Surgeons, Fellowship in the Orthopedic Section of the New York Academy of Medicine, may be accepted as evidence of qualification.

Physicians who have passed the examinations of the American Board of Orthopedic Surgery may be accepted provided they conform to the requirements of years of experience in orthopedic surgery.

Requirements for Rating in Roentgenology

HIGH ethical and professional standing.
2. The applicant must be a physician licensed to practice medicine in the State of New York and currently registered with the State Department of Education.

3. The applicant for a rating in radiology shall submit evidence that his office or the hospital department with which he is connected is properly and adequately equipped for the practice of radiology.

4. He shall submit evidence of training and experience in the field of roentgenology subsequent to graduation from medical school. This training should have been obtained as a post-graduate student in an acceptable college or hospital as part of a regularly conducted course in roentgenology over a period of one or two years, followed by association with an acceptable voluntary or muncipal hospital for a period of one to three years.

Address at a Dinner of the ASSOCIATION FOR THE ADVANCEMENT OF INDUSTRIAL MEDICINE AND SURGERY, Hotel New Yorker, New York City, November 28, 1940.

5. The Committee may accept as evidence of qualification a position as roentgenologist on the x-ray staff of a voluntary or municipal hospital and may waive the post-graduate requirements for a physician who has had such a position for a period of three years or more. The Committee must be convinced that the activity of the x-ray department is sufficient to permit the roentgenologist to become really experienced.

6. The Committee will accept as evidence of qualification the diploma of the American Board of Radiology.

7. If the Committee is in doubt as to a physician's qualifications in roentgenology or radiation therapy, he may be given an examination in roentgenology or radiation therapy or both by a special examining committee appointed by the State Medical Society and if he passes such examination shall be qualified in roentgenology provided he meets the requirements in respect to equipment.

8. Physicians who confine their work to radiology and who meet the above requirements shall be given a rating of SD. Physicians who do not confine their work to roentgenology but who also meet the above requirements, shall be given the rating XD. The D rating shall not be given to a physician engaged in any specialty, such as surgery, orthopedic surgery, gastro-enterology, etc., solely because of expertness in x-ray examination or interpretation within his specialty. A physician shall receive the D rating only if he is *generally* qualified in the field of roentgenology.

The D-1 rating denotes general and full qualification in the field of diagnostic radiology or roentgenology. The D-2 rating denotes general qualification in the field of radiation therapy. The D rating denotes qualification in both fields.

Requirements for Rating in Ophthalmology

A CANDIDATE for qualification in the field of ophthalmology shall present a license to practice medicine in the State of New York, and evidence of registration for the current year with the Department of Education.

2. The candidate shall be of high ethical and moral standing in the profession.

3. The Committee may require letters of recommendation from outstanding physicians and specialists in the field of ophthalmology in support of the above and as evidence of professional standing.

4. The applicant shall present evidence of a regular internship of from one to three years in a duly accredited hospital and shall also present evidence of a residency in the specialty of ophthalmology covering a period of from one to three years, in an institution recognized as qualified to teach residents in this specialty or the equivalent of this training.

5. Subsequent to the completion of a residency, the physician shall have had experience in private practice, and in hospital or clinical affiliations, for a period of at least one year; this being dependent on the length of training in the specialty during internship and residency. Where the period of residency has been two to three years, the minimum period of private practice may be accepted. Where the residency has been of but one year following internship, a minimum of at least three years of subsequent training in the specialty shall be required. Physicians who have been in private practice for 10 years or more and have not had a period of residency in the specialty, but have been appointed to positions on the attending staff of a reputable and accredited hospital in the specialty may be qualified provided they can produce satisfactory evidence of independent ophthalmological (surgical) work during this period of hospital association.

6. In the case of reputable well-known specialists of outstanding ability any or all of the above standards may be waived by the Committee.

7. The diploma of the American Board of Ophthalmology may be accepted in lieu of evidence of internship and residency provided, however, the possessor of such diploma shall have had at least one year of experience in private practice. In the case of physicians trained in foreign countries who are not of outstanding ability and reputation or whose credentials may be difficult to substantiate, the Committee should require evidence of association with an accredited institution in this country for a period of at least one to three years together with letters of recommendation from local specialists connected with such institution and known to the Committee.

8. The specialist designation "SE" shall be granted only to such physicians as meet the above requirements and who have been graduated from a medical school at least seven years previously. (This may be modified in small communities where the specialist will be expected to do at least 75% of special work.)

9. Physicians may be granted the "XE" qualification who meet the above requirements after five years following graduation from medical school during which period the applicant shall have had sufficient training in ophthalmology in hospital clinic or private work.

The "XE" qualication may be granted to physicians who do some general practice, but whose major interest is in the specialty and who have not had sufficient experience to be granted a full specialist's rating.

10. The qualifying Committee has the right to request an applicant for specialist rating or for a special qualification in the field to appear personally before the Committee to support his application for qualification.

11. The Committee also has the right to request the applicant to submit a certified list of operative procedures performed independently by the applicant during the specified period.

Laryngology, Rhinology and Otology

A CANDIDATE for qualification in the field of Oto-laryngology shall present a license to practice medicine in the State of New York and evidence of registration for the current year with the Department of Education.

2. The candidate shall be of high ethical and moral standing in the profession.

3. The Committee may require letters of recommendation from outstanding physicians and specialists in the field of oto-laryngology in support of the above and as evidence of professional standing.

4. The applicant shall present evidence of a regular internship of from one to three years in a duly accredited hospital and shall also present evidence of a residency in the speciality of oto-laryngology covering a period of from one to three years, in an institution recognized as qualified to teach residents in this specialty.

5. Subsequent to the completion of a residency the physician shall have had experience in private practice and hospital or clinical affiliations for a period of at least one year, this being dependent on the length of training in the specialty during internship and residency. Where the period of residency has been two to three years, the minimum period of private practice may be accepted. Where the residency has been of but one year following internship, a minimum of at least three years of subsequent training in the specialty may be required. Physicians who have been in private practice for 10 years or more and have not had a period of residency, in the specialty, but have been appointed to positions on the attending staff of a reputable and accredited hospital in the specialty may be qualified provided they can produce satisfactory evidence of independent oto-laryngological (surgical) work during this period of hospital association.

6. In the case of reputable well-known specialists of outstanding ability any or all of the above standards may be waived by the Committee.

7. The diploma of the American Board of Oto-Laryngology may be accepted in lieu of evidence of internship and residency provided however the possessor of such diploma shall have had at least one year of experience in private practice. In the case of physicians trained in foreign countries who are not of outstanding ability and reputation or whose credentials may be difficult to substantiate, the Committee should require evidence of association with an accredited institution in this country for a period of at least one to three years together with letters of recommendation from local specialists connected with such institution and known to the members of the Committee.

8. The specialist designation "SF" shall be granted

only to such physicians as meet the above requirements and who have been graduated from a medical school at least seven years previously. (This may be modified in small communities where the specialists will be expected to do at least 75% of special work.)

9. Physicians may be granted the "XF" qualification who meet the above requirements after five years following graduation from medical school during which the applicant shall have had continuous and active association in this specialty in hospital or clinic work.

10. The rating "XF" may be granted to qualified physicians who do some general practice, but whose major interest is in the specialty and have not had sufficient experience to be granted a full specialist's rating.

11. The qualifying committee has the right to request an applicant for specialist rating or for a special qualification in the field to appear personally before the Committee to support his application for qualification.

12. The Committee also has the right to request an applicant to submit a certified list of operative procedures performed by him independently during a specified period.

Dermatology and Syphilology for both SH and XH

HIGH ethical and professional standing.

2. The applicant must be a physician licensed to practice medicine in the State of New York and registered for the current year with the State Department of Education.

3. Satisfactory completion of a general internship of at least one year.

4. Letters of recommendation from two well known dermatologists.

5. Satisfactory training in dermatology and syphilology as mentioned in the following paragraph:

(a) *For SH* applicants shall have qualifications equal to those which give permission to take the examination of the American Board of Dermatology and Syphilology. This includes two years of graduate training in dermatology and syphilology (as outlined in the booklet published by the Board) plus three years of practice limited to this specialty. For those who begin their training after January 1, 1940, three years of intensive training, plus two years of practice limited to this specialty will be required. As an alternative if an applicant has held an appointment for 10 years or more in a well equipped service of dermatology and syphilology of a recognized hospital, he shall qualify for SH rating, provided his practice is limited to dermatology and syphilology. An applicant who qualifies for SH rating shall have confined his (or her) entire professional time for at least five years to the study and practice of dermatology.

(b) *For XH* an applicant who has had sufficient training in dermatology and syphilology (as aforementioned for applicants for SH rating) but (1) whose practice is not confined exclusively to this specialty or (2) who lacks the necessary years (five) of practice in this specialty, may be entitled to a rating of XH.

Neurology and Psychiatry

A CANDIDATE for qualification in the field of neurology and psychiatry shall present a license to practice medicine in the State of New York and evidence of registration for the current year with the Department of Education.

2. Evidence of at least one year general internship in an approved hospital.

3. At least three years' resident training in neurology and psychiatry in an approved hospital or hospitals devoted to neurology and/or psychiatry or in a general hospital having a service devoted to these specialties. The training is to include six months or more in neuroanatomy, neurophysiology, and neuropathology.

4. A period of at least three years in private practice during which the applicant shall have had a position on the attending staff of a recognized neurologic or psychiatric service of a special or general hospital.

5. The diploma of the American Board of Neurology and Psychiatry may be accepted as evidence of qualification in this field.

In exceptional cases the Committee may accept other qualifications which they deem as the equivalent of those stated above.

Gynecology and Obstetrics

HIGH ethical and professional standing.

2. The applicant must be a physician licensed to practice medicine in the State of New York and registered for the current year with the State Department of Education.

3. Certification by the American Board of Obstetrics and Gynecology will be acceptable as evidence of qualification.

4. A position as consulting gynecologist or obstetrician or as full attending or associate visiting gynecologist or obstetrician on a duly constituted staff of a Grade A hospital.

5. A position as adjunct visiting gynecologist or obstetrician on the staff of a duly constituted Grade A hospital of at least five years' standing.

6. A position of attending or associate visiting surgeon in a Grade A hospital where the gynecological cases are under the supervision of the general surgical service, and where the applicant is in active charge of gynecological or obstetrical cases.

7. Physicians who have had training in gynecology or obstetrics as evidenced by a two-year general surgical internship in an acceptable hospital followed by a residency of at least two years in gynecology and obstetrics, with at least one year in private practice in the specialty.

8. Physicians who have had a general surgical internship followed by a residency in gynecology and obstetrics and who have had at least two years' experience in private practice in this specialty.

9. Certain physicians having exceptional training in gynecology and obstetrics not covered by the above may be qualified by the Board if they can give proof of such adequate training and experience in the fields of gynecology and obstetrics acceptable to the Board and in consonance with the spirit of the above requirements.

Requirements in Physical Therapy (M-1)

HIGH ethical and professional standing.

2. The applicant must be a physician licensed to practice medicine in the State of New York and currently registered with the State Department of Education.

3. Completion of an internship and residency in a general or special hospital, of one to two years, together with additional evidence of special training in hospital or dispensary work and in private practice for at least one to three years.

4. Evidence of special training in physical therapy followed by clinical work in physical therapy extending over at least five years in an accepted department of physical therapy; or in private practice for at least 10 years.

5. The Board may accept in lieu of the above evidence of special ability by either teaching or writing or by passing a qualifying examination before a special examination board.

THESE standards have been distributed throughout the state and are being used by the County Societies' compensation boards and committees in qualifying physicians. It must be borne in mind that while the standards are more or less rigidly adhered to in the larger counties where there are numerous physicians who confine their practices to a given specialty, in certain of the rural counties it is not always possible to adhere to the very high standards that apply in the Metropolitan area, and other big cities of the state. It is the right of the local county society's compensation boards so to modify these standards as to conform to the actual practice in any given community. In other words, in the small communities where a physician is generally regarded as a specialist or consultant in private practice and is especially qualified, he is so designated

under the Workmen's Compensation Law, even though he may not devote himself 100% to the field in which he is especially qualified.

It is the function of the local compensation committee to verify the credentials submitted by the applicant physician. It is also their function and responsibility to obtain confirmatory evidence from hospitals and from other practitioners as to the applicant's standing and capabilities. A diploma or certificate of the National Specialty Board is usually accepted as prima facie evidence of qualification in a specialty, since these diplomas are issued only after examination in the specialty at this time. In the field of x-ray, the State Medical Society has set up a special examining committee. Applicants for x-ray rating who do not possess the diploma of the American Board of Radiology, and who cannot satisfy the local county society committee of their qualifications, are referred for examination to this committee. A practical and oral examination is given about four times a year and a roentgenological qualification recommended if the applicant passes this examination. It must also be borne in mind that under present chaotic world conditions, it is very difficult to verify and evaluate the credentials of refugee physicians. Where these physicians apply for a specialty rating, unless they can satisfy the local boards as to their qualifications, usually by letters from New York physicians who are familiar with their standing abroad and can personally vouch for them, it is the policy of most of the boards to give a general rating and await an opportunity to evaluate the refugees' qualifications by personal observation of their work in accredited institutions in this state. This has worked out very well and without prejudice. In certain instances it has been necessary to appoint a committee of surgeons or specialists to observe the actual work of the refugee physicians, (as well as graduates of United States schools), on patients in New York hospitals. It should be borne in mind that both the standards and methods of qualifying physicians have evolved as the result of experiences since the inception of the new law in 1935. While perfection has not been reached, I would like to point out that the procedure is working with great efficiency and promptness and with justice to the applicant, and to the end that the injured workmen of this state may receive the best quality of medical care.

It may be of interest to know the number of physicians authorized to treat Workmen's Compensation cases to date.

There are 19,421 physicians authorized to treat Workmen's Compensation cases in this state — 13,248 practicing in the New York District, 1,829 in the Albany District, 1,447 in the Syracuse District, 1,146 in the Rochester District, 1,398 in the Buffalo District, and 353 Osteopaths.

From these figures you will note that nearly all the active practising physicians in the state are authorized to do compensation work, as I understand there are about 22,000 physicians licensed to practice in the state.

Historical Milestones

—*in Occupational Medicine and Surgery*—

ALFRED H. WHITTAKER, M.D., F.A.C.S.,
and
DAVID J. SOBIN, M.D., M.S.,
Detroit

SEVENTEEN years before Columbus made his historic discovery in the search for a new route to the Indies, ULRICH ELLENBOG was troubling his mind with the occupational hazards of the goldsmiths and other workers in metals. He had already written the manuscript and was searching for a method to spread his message when opportunity rapped on his door. A need had arisen in Augsburg for someone to sponsor the new-fangled printing press. Ellenbog saw his chance and helped to found the press of the Abbey of SS. Ulrich and Afra in that city. For the 60 years since Gutenberg's invention, or until about 1500, books resembled the old manuscripts in form and content; but with the founding of the press at Augsburg books took on a more popular character, were published in the everyday vernacular, and were intended for the lay public. This change in approach heralded the end of the Middle Ages, but it is difficult to state with certainty where and when this period terminated as different countries and different provinces accepted the changes with different degrees of alacrity; however, in general, the end of the incunabula marks the beginning of the Renaissance.

ULRICH ELLENBROG was born at Feldkirch, Austria in 1440, and died at Memmingen in 1499. He was a Doctor of Medicine, and physician to the Bishop and Chapter of the Augsburg Cathedral from 1470 to 1478. It was during these few years that the press of the Abbey of SS. Ulrich and Afra was founded in Augsburg under his direction. In 1473 he wrote the manuscript of the book described below,* which was published posthumously a half century later. He wrote several other medical books and possessed a large library which was still extant, in part, at the opening of the World War II. He bequeathed his books and unpublished manuscripts to his son, Nicolaus, a Benedictine Monk at Ottobeuren, where some of his books are still preserved. Only three of Ellenbog's manuscripts were ever published, among which is the booklet at hand, which was formerly thought to have been printed after the sack of the Ottobeuren Library during the peasant uprising in 1524.

Of Ellenbog's booklet, Dr. C. F. Mayer, Librarian of the Army Medical Library, has this to say:

"The type of letters in Ellenbog's work was the chief evidence that the book was printed by Melchior Ramminger, of Augsburg. The date of publication has been determined with the aid of the curious title page of which you sent me a photograph. This cut, as I now know definitely,

* The first printed thesis dealing with occupational health—there being two copies extant, of which this one is in the library of the senior author, the other in the University Library, Munich.

represents a shoemaker and cannon, and was first used by the printer in 1523 as the title page of a book written by Hans Sachs, the shoemaker poet, and called: DISPUTATION ZWICHEN EINEM CHORHERREN UND SCHUCHMACHER DARIN DAS WORT GOTTES UND EINRECHT CHRISTLICH WESEN VERFOCHTEN WURDT. I am still unable to tell who 'V. Engelshofen' is; the three 'sevens' in the lower right hand corner have no special meaning and serve possibly the purpose of balancing the page. The written figure *1533* may be somebody's guess as to the date of printing, which as I mentioned, has been determined by circumstantial evidence and accepted to be between 1523 and 1524."

This little eight-page booklet was first translated by C. Barnard and published in the Lancet, 1932 (p. 270), however, the numerous minor discrepancies between his translation and our own makes it interesting to publish Dr. Ernst's version. The differences in translation are due to the fact that the booklet was published in the vernacular of the day, that many of the words are no longer in the dictionary, and that even some of the letters are no longer used. An analogy would be to publish Beowulf or Chaucer in the original version and ask a medical student to read it.

*CONCERNING THE POISONOUS, EVIL VAPOURS AND FUMES OF METALS, SUCH AS SILVER, QUICKSILVER, LEAD AND OTHERS WHICH THE WORTHY TRADE OF THE GOLDSMITH AND OTHER WORKERS ARE COMPELLED TO USE. HOW THEY MUST CONDUCT THEMSELVES CONCERNING THESE MATTERS, AND HOW TO DISPEL THE POISON.

TO THE ROYAL SUBSIDIARIES AND WORTHY TRADE OF GOLDSMITHS OF THE IMPERIAL CITY OF AUGSBURG.

My voluntary and conscientious service shall be ready for you all. Since I, Ulrich Ellenbog, Doctor of Medicine at Feldkirch, at present chief physician of the Exalted Chapter at the Augsburg Cathedral, have considered the great and highly injurious harm which faces this master trade through the smoke and fumes of the materials with which the tradesman is compelled to work. Therefore I do not wish to withdraw myself and remain aloof to the masters of this trade, but to serve them by giving assistance and advice in order that they may not receive too severe an illness which is produced by these fumes.

I beg of you with all my heart to take this, my modest advice, written with my own hand in all good will and friendship. Nothing more would I desire than to serve each and all with friendly service. Dated: August 6, 1473

CONCERNING COAL

Modern manufacturing plants are using coal for fuel, the smoke and fumes of which are poisonous, especially when the coal is somewhat damp. When these vapours are heavy and accumulate above the

Von den giftigen Besen Tempffen
vñ Reüchen, der Metal/als Silber, Queck-
silber Bley vnd anders So die Edlen hand
werck des Goltschmidens/vnd ander arbai-
ter in des feür sich gebrauchen müssen Wie sie
sich da mit halten vnd die gift vertreibē sollē.
V. Engelshofen.
1533.
777

Concerning poisons and the evil vapours of smoke and of metals, such as silver, quicksilver, lead, and others which the worthy trade of the goldsmith and other workers are compelled to use. How they should conduct themselves concerning these matters and how to dispel the poison.

coal they explode. When a person loiters near this fire for a period of time his countenance becomes pallid, so that he appears green and blue, his eyes dim, and he struggles for air. This vapour causes a heavy unnatural sleep, and at times heavy limbs, and if one tarries a longer period of time he becomes palsied.

All this does not require much damp coal for it will burn just as well in the open air. In the winter time, if you have a fire of coal in the shop or factory, then do not close the windows but guard yourself against the vapours as much as possible. This is my advice. The workers reveal that they are fully poisoned and feverish when they live and sleep a long time in an enclosure where there is coal vapour, as has been proved by some leaders in Medicine. When the employers or masters demand that coal fires be lit, then blow them up in the open air and lay white incense upon it or sprinkle a little wine over it, for this causes the poison and the danger of the vapour to be diminished.

* Acknowledgement and thanks is herewith tendered to Reverend John L. Ernst, Superintendent of the Deaconess Hospital, Detroit, for his help in translating this volume.

Concerning the Vapours of Quicksilver, Lead and Other Metals with which the Goldsmith Ordinarily Labours

The profession of Medicine holds that the vapour of every metal retains the natural complexion and power of the kind from which it arises. The pharmacists usually prepare distilled water of a warm nature if they derive them from warm herbs. Accordingly they also believe that the vapour and smoke of each metal retains the natural complexion and power from which it arises.

Now it is true that oftentimes the vapours and smoke are more powerful than the metal or mineral, as it appears when one sublimates aquafortis, also when one distils brandy for there the vapour is much stronger than the ammonia alone or the wine wherefrom the brandy is made. Therefore men call the vapour spirits, that is ghosts for through their appearance and strength they always work more strongly than the body from which they are drawn. Now it is obvious in natural history that the nature of quicksilver is rather cooling and moist in appearance and action, lead also is cooling and moist, the same is true of silver, it is of a cool nature; likewise antimony and litharge are cold. Wherefore I state that the smoke and vapour of such metals chill greatly the man who has to work therewith. Therefore when ye masters and men work with lead, and when ye gild, guard yourself against the vapours and smoke for it is poisonous to you. This, my advice, is that they should be kept in the open air as much as possible, and never in a tight enclosure. Workers should not hold their heads above this vapour but should wear a mask. The vapour of quicksilver, silver, and lead are a cooling poison, for they make a heaviness and tightness in the chest, burden the limbs and often lames them, as one can often see in the foundries where men work with large quantities of these metals.

When one Wishes to Reduce the Harm or Injury of These Vapours

When you are engaged in the filing or plating of metal have musk by you and smell of this much and often, for the vapour of the musk by its warmth supresses the coldness of the smoke of lead, silver, and quicksilver. Also the musk, by virtue of the property of strengthening the heart and brain, wards off and assuages the harm from the vapour of lead and quicksilver; thus these vapours cannot harm you.

Likewise, any time you goldsmiths linger in the aforesaid fumes, keep in your mouth one of the following pieces, which ye will, and chew it so that by the strength of these vapours will be corrected and lessened the strength of the vapour that you draw in with your breath. Thus these vapours will not harm you, as they would did you not keep these pieces in your mouth. And these are the pieces (*antidotes*), juniper berries, white root of torimetal, elecampane, rue, dittany (and others). Many other pieces are commended by the physicians against these poisonous vapours, but these aforesaid are good for you, and common enough so that you can get them at slight cost. Several teachers of Medicine highly commend the emerald, which one shall also hold in the mouth, for it withstands the poisoning by these vapours. Also, dear craftsmen, if one is gilding or plating, and has inhaled much vapour so that he feels distressed and fears harm, then he shall on the following day, on a fasting stomach, perspire in bed and then take a good old theriac, the size of a large hazel nut, with wine; thus will the poison which has come into him through his work with this metal be driven from his system. The doctors also recommend that those working with metals or gilding should drink vermouth wine, through the heat of which will the evil chill of the vapours be depressed, and whoever has no vermouth in his cellar may in a day or seven hours make it by dropping wormwood into wine. If however, one cannot perspire then he should go on an empty stomach to the bathroom after having taken the theriac mentioned above.

Concerning the Vapours of Aquafortis which are warm, and very sharp, and against the vapours of litharge and antimony, which are of a cold nature, you shall guard especially well, as is described above in connection with lead and quicksilver.

Against the Vapours of Verdigris, Saltpeter and Sal Ammoniac you must be very careful and guard yourself while working with pale gold, for these vapours are very sharp and poisonous. To correct and lessen their poison one should drink half a dram of bole mixed with wine and rose water. Herewith God keep us all in good health. Amen.

The Pittsburgh Meeting

—*Institute of Industrial Medicine and Industrial Hygiene*—

FOLLOWING is the complete program of the annual meetings to be held in Pittsburgh, Pennsylvania, May 5, 6, 7, 8, and 9, 1941 — the Twenty-Sixth Annual Meeting of the American Association of Industrial Physicians and Surgeons, and the Second Annual Meeting of the American Industrial Hygiene Association:

Monday, May 5

REGISTRATION—No Fee
9:00–10:00—William Penn Hotel
LECTURES AND DEMONSTRATIONS
Mercy Hospital, Stevenson and Locust Street, Pittsburgh
John P. Griffith, M.D., *Chairman*
10:00–12:30—Mercy Hospital Staff
12:30– 1:30—Lunch
1:30– 4:30—Mercy Hospital Staff
Monday will be devoted to a series of Dry Clinics in Industrial Medicine and Surgery at Mercy Hospital.

Tuesday Morning, May 6

9:00—ADDRESS OF WELCOME
Hon. Cornelius D. Scully, *Mayor, Pittsburgh.*

9:15—ADDRESS OF WELCOME
FREDERICK JACOBS, M.D., *President, Allegheny County Medical Society*
9:25—ADDRESS OF WELCOME
JOHN T. SHAW, M.D., *Secretary of Health, Commonwealth of Pennsylvania*
9:35—RESPONSE
DANIEL L. LYNCH, M.D., *President of the American Association of Industrial Physicians and Surgeons*
9:40—RESPONSE
MR. WARREN A. COOK, *President of the American Industrial Hygiene Association*

ANNUAL MEETING—AMERICAN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS

9:45–10:10 President's Address, DANIEL L. LYNCH, M.D.
10:10–10:30 "Some Practical Results of Periodic Physical Examinations"—A. J. KAMMER, M.D., *Medical Director, Inland Steel Company,* Indiana Harbor, Indiana
10:30–10:40 Discussion
10:40–11:00 "Relation of Trauma to Disease"—A. H. COLWELL, M.D., *Associate Professor of Medicine, School of Medicine, University of Pittsburgh*
11:00–11:10 Discussion
11:10–11:40 "Contact Dermatoses"—W. H. GUY, M.D., *Professor of Dermatology, School of Medicine, University of Pittsburgh*
11:40–12:00 Discussion
12:30– 2:00 Luncheon and Exhibits

Tuesday Afternoon, May 6

2:00– 2:20 "Sequelæ of Head Injuries"—S. S. ALLEN, M.D., Pittsburgh
2:20– 2:30 Discussion
2:30– 2:50 "Trauma Other Than Fractures"—J. H. WAGNER, M.D., *Carnegie-Illinois Steel Corporation,* Pittsburgh
2:50– 3:00 Discussion
3:00– 3:20 "Mental Hygiene in Industry"—C. H. HENNINGER, M.D., *Professor of Psychiatry, School of Medicine, University of Pittsburgh*
3:20– 3:30 Discussion
3:30– 3:50 "Evaluation of Circulatory Function in Extremities"—M. B. FERDERBER, M.D., *Department of Industrial Hygiene, School of Medicine, University of Pittsburgh*
3:50– 4:00 Discussion
4:00– 4:30 Business Session
4:30– 6:00 Exhibits

Tuesday Evening, May 6

INDIVIDUAL GROUP CONFERENCES

Wednesday Morning, May 7

9:00– 9:20 "Medical and Preventive Aspects of Carbon Monoxide Poisoning"—F. S. ROSSITER, M.D., *Carnegie-Illinois Steel Corporation,* Pittsburgh
9:20– 9:30 Discussion
9:30–10:00 "Recent Investigations of Effects of Radiation from an Ophthalmologic Viewpoint"—C. F. KUTSCHER, M.D., Pittsburgh
10:05–10:35 "The Absorption and Excretion of Ingested Lead Under Various Experimental Conditions"—R. A. KEHOE, M.D., *Kettering Laboratory of Applied Physiology, University of Cincinnati,* Cincinnati, Ohio
10:45–11:05 "Engineering Control of Lead Contaminated Environments"—G. C. HARROLD, Ph.D., *Industrial Hygiene Laboratories, Chrysler Corporation,* Detroit, Michigan
11:10–11:25 "Lead Exposures in the Printing Industry"—R. T. HOMEWOOD, *Virginia Department of Health,* Richmond, Va.
11:30–11:45 "Incidence of Lead Poisoning in the City of Baltimore"—E. KAPLAND, Ph.D. and J. M. McDONALD, M.D., *Baltimore Health Department,* Baltimore, Md.
11:45–12:00 Discussion
12:00– 2:00 Luncheon and Exhibits

Wednesday Afternoon, May 7

2:00– 2.20 "Tuberculosis and Its Allied Conditions as Commonly Seen in Our Industrial Population"—C. H. MARCY, M.D., *Director Tuberculosis League Hospital,* Pittsburgh
2:20– 2:40 "Relationship between Tuberculosis and Inhalation of Various Substances from a Pathological Standpoint, Including Bituminous Coal Dust Inhalation"—S. R. HAYTHORN, M.D., *Director Singer Laboratory, Allegheny General Hospital,* Pittsburgh
2:40– 2:50 Discussion, R. R. SAYERS, M.D., *Director U. S. Bureau of Mines,* Washington, D. C.
2:55– 3:20 "Engineering Preventive Methods in the Control of Tuberculosis"—D. E. CUMMINGS, *Director Division of Industrial Hygiene, University of Colorado,* Denver, Colorado
3:25– 3:45 "Control of Tuberculosis and the Employment of a Tuberculous Individual in Industry"—W. A. SAWYER, M.D., *Medical Director, Eastman Kodak Company,* Rochester, N. Y.
3:45– 3:55 Discussion
3:55– 4:20 "Bacteria in Air and Disinfection"—A. E. WILLIAMSON and H. B. GOTAAS, *Department of Sanitary Engineering, School of Public Health, University of North Carolina,* Chapel Hill, N. C.
4:20– 4:35 "Variation in Bacterial Population in Certain Ventilating Systems"—A. HOLLAENDER, Ph.D., *National Institute of Health,* Bethesda, Md.
4:35– 4:45 Discussion
4:45– 6:00 Exhibits

Wednesday Evening, May 7

6:30—BANQUET

"The Role of Industrial Medicine and Industrial Hygiene in National Defense"—IRVIN ABELL, M.D., *Chairman of Health and Medicine Committee, Federal Security Agency,* Washington, D. C.

"Noise and Hearing"—V. O. KNUDSEN, Ph.D., *Dean of the Graduate School, University of California,* Berkeley, California
WESTINGHOUSE QUARTET

Thursday Morning, May 8

8:30– 8:50 "Design of Lateral Exhaust Hoods for Industrial Gases, Vapors and Fumes"—L. SILVERMAN, *Harvard School of Public Health,* Boston, Mass.
8:55– 9:15 "Ventilating for Electroplating Operations"—A. D. BRANDT, Sc.D,. *National Institute of Health, Division of Industrial Hygiene and Sanitation,* Bethesda, Md.
9:15– 9:25 Discussion
9:25– 9:50 "Airborne Infection and Disinfection in Air Conditioning"—W. F. WELLS, *University of Pennsylvania,* Philadelphia, Pa.
9:55–10:20 "Local Cooling of Workers in Hot Atmospheres"—F. C. HOUGHTEN and M. B. FERDERBER, M.D., *American Society Heating and Ventilating sylvania,* Philadelphia
10:25–10:50 "Radiant Cooling"—C. P. YAGLOU and O. SCHNEIDER, PH.D., *Harvard School of Public Health,* Boston, Mass.
10:50–11:05 Discussion
11:05–11:20 "Comparison of Quick Methods for Quantitation of Impinger Samples of Granite Dust"—C. L. POOLE, *Rhode Island State Department of Health,* Providence, R. I.
11:25–11:40 "An Immersion Method for the Rapid Approximation of Quartz in Granite"—W. B. WARDLOW, *Texas State Board of Health,* Austin, Tex.
11:45–12:00 "Use of X-ray Diffraction Equipment to Determine Silica in Dust Exposures"—J. W. BALLARD,

Ph.D., *U. S. Bureau of Mines, Pittsburgh Experiment Station,* Pittsburgh
12:00–12:15 General Discussion
12:15– 1:30 Luncheon and Exhibits

Thursday Afternoon, May 8

1:30– 1:45 "The Effects of the Inhalation of Dusts Encountered in the Chrome Brick Industry"—W. B. FULTON, M.D., and F. E. BUTTERS, *Division of Industrial Hygiene, Pennsylvania Department of Health,* Harrisburg, Pa.
1:50– 2:05 "Engineering Survey in Tri-State Lead and Zinc Mining District"—E. C. WARKENTIN, *Oklahoma State Department of Health, Division of Industrial Hygiene,* Oklahoma City, Okla.
2:10– 2:25 "Industrial Hygiene Control on Tunnel Construction in Hydro-Electric Projects in Western North Carolina"—M. F. TRICE, *Industrial Hygiene Engineer, Division of Industrial Hygiene, North Carolina State Board of Health,* Raleigh
2:25– 2:40 Discussion
2:40– 2:55 "Etiological Aspects of Particle Size in Quartz Dust" — B. D. TEBBENS, and R. SCHULTZ, M.D., *Harvard School of Public Health,* Boston, Mass.
3:00– 3:20 "Particle Sizes of Dust as Found in Various Industries and Determination of Silica in Bituminous Coal Dusts"—C. E. BROWN, Sc.D., and H. H. SCHRENK, PH.D., *U. S. Bureau of Mines, Pittsburgh Experiment Station,* Pittsburgh
3:20– 3:30 Discussion
3:30– 3:45 "Animal and Petrographic Studies on Talc Dusts"—C. WILLIAMS, PH.D., *Liberty Mutual Ins. Co.,* and R. SCHULTZ, M.D., *Harvard School of Public Health,* Boston, Mass.
3:50– 4:05 "Fuller's Earth as a Cause of Pneumoconiosis"—W. D. MCNALLY, M.D., *Consulting Toxicologist,* Chicago
4:10– 4:35 "Silicosis Among Naval Foundry Men"—E. W. BROWN, M.D., and W. E. KLEIN, M.D., *Medical Corps, U. S Navy*
4:35– 5:00 Discussion
5:00– 6:00 Exhibits

Thursday Evening, May 8

7:30– 8:00 "Review of Industrial Hygiene Progress During 1940"—WARREN A. COOK, *President, American Industrial Hygiene Association*
8:00– 8:15 "Claims of Industrial Poisoning"—P. N. HARGER, Ph.D., *Prof. of Biochemistry and Toxicology, University of Indiana, School of Medicine,* Indianapolis, Ind.
8:20– 8:35 "Carbon Disulfide and Hydrogen Sulfide Exposure in the Synthetic Sausage and Casing Industry using the Viscose Process"—MILTON H. KRONENBERG, M.D., KENNETH MORSE, M.S., ALFRED SETTERLIND, M.S., *Division of Industrial Hygiene, Illinois Department of Health,* Chicago
8:40– 8:55 "Sampling and Determination of Aldehydes"—E. C. BARNES, *Industrial Hygiene Laboratory, Westinghouse Electric and Manufacturing Company,* East Pittsburgh, Pa.
9:00– 9:25 "The Absorption and Elimination of Noxious Vapors"—H. W. HAGGARD, M.D., *Director of Laboratory of Applied Physiology, Yale University,* New Haven, Conn.
9:25 Discussion

Friday Morning, May 9

8:30– 8:45 "Dermatitis Control in a Mass Production Industry"—T. F. MOONEY, *Ford Motor Co.,* Dearborn, Mich.
8:50– 9:05 "Sporotrichosis as an Occupational Disease Among Florists"—L. W. SPOLYAR, M.D., *Indiana State Board of Health, Industrial Hygiene Division,* Indianapolis, Ind.
9:10– 9:35 "A Statistical Approach in the Study of Hematologic Data from Some Common Solvent Exposures"—J. H. STERNER, M.D., *Eastman Kodak Co.,* Rochester, N. Y.
9:40– 9:55 "Recovery Following Exposure to Benzol"—L. J. GOLDWATER, M.D., *New York University, Dept. of Preventive Medicine,* New York City.
9:55–10:10 Discussion
10:10–10:30 "Electric Arc Welding, Coated Rods"—C. P. MCCORD, M.D., S. F. MEEK, M.D., and G. C. HARROLD, Ph.D., *Industrial Hygiene Laboratories, Chrysler Corporation,* Detroit, Mich.
10:35–10:55 "Gases, Fumes and Vapors from Coated Welding Rods"—B. D. TEBBENS, Sc.D., and PHILIP DRINKER, *Harvard School of Public Health,* Boston.
10:55–11:05 Discussion
11:05–11:25 "Trends in Occupational Disease and Industrial Hygiene"—H. B. MELLER, Sc.D., and F. S. MALLETTE, *Air Hygiene Foundation, Mellon Institute,* Pittsburgh
11:30–11:50 "The Human Eye and Its Protection in Industry"—W. H. LEHMBERG, Sc.D., *American Optical Company,* Southbridge, Mass.
11:50–12:00 Discussion
12:00– 1:30 Exhibits

Friday Afternoon, May 9

ANNUAL MEETING, SECOND SESSION
1:30– 1:50 Election of Officers
1:55– 2:10 "Controlled Radium Hazards" — FREDERICK FLINN, Ph.D., *Columbia University,* New York City
2:15– 2:35 "Some Recent Observations on Phosphorus and on Ammonium Nitrate Toxicology"—R. B. L. FLEMING, *Tennessee Valley Authority*
2:40– 2:55 "Biochemistry of Carbon Bisulfide"—R. W. MCKEY, PH.D., *Harvard School of Public Health,* Boston, Mass.
2:55– 3:10 Discussion
3:15– 3:30 "Toxicity of 1-1 Dichlor-1-nitroethane"—WILLARD MACHLE, M.D., *Kettering Laboratory of Applied Physiology, University of Cincinnati,* Cincinnati, Ohio
3:35– 3:50 "Acute Toxic Effects Upon Experimental Animals of Methyl, Ethyl and Butyl Methacrylates"—WILHELM DEICHMANN, Ph.D., *Kettering Laboratory of Applied Physiology, University of Cincinnati,* Cincinnati, Ohio
3:55– 4:10 "Toxicity of Methyl Silicate"—W. G. FREDRICK, Ph.D., *Industrial Hygiene Bureau,* Detroit.
4:15– 4:30 Discussion
4:30– Business Meeting (Annual)

American Association
of Industrial Physicians and Surgeons

—Constitution and By-Law Amendments Proposed to be Voted upon at the Annual Meeting, May 5-9, 1941—

CONSTITUTION:

ARTICLE V, SECTION 3: After the words "twelve directors" in line 4, insert "and a House of Delegates consisting of the Officers and members of the Board of Directors and the Presidents or legally appointed or elected delegates of Component Societies." Delete that part of Section 3 beginning with "Presidents of Component Societies" and ending with "as other directors."

ARTICLE V: Change heading to read "Officers, Directors and Governing Bodies."

ARTICLE V, SECTION 1: After the words "Second Vice-President" insert "a Managing Director" and delete the word "Secretary" in the compound word "Secretary-Treasurer."

ARTICLE V, SECTION 4: Delete entire section which will be placed in the By-Laws.

ARTICLE V, SECTION 5 becomes Section 4, and change the word "five" to "six."

BY- LAWS:

ARTICLE I, SECTION 2: Change it to read "Special meeting of the membership may be called by the President with the approval of five members of the Board of Directors. Special meetings of the Board of Directors or of the House of Delegates may be called at the discretion of the President."

ARTICLE I, SECTION 3: Insert after the words "all meetings" the words "of the members" and delete the word "Treasurer" in the compound word "Secretary-Treasurer."

ARTICLE I, SECTION 4: Delete the word "Treasurer" in the compound word "Secretary-Treasurer."

ARTICLE IV: Change heading to "Duties of Officers and Governing Bodies."

ARTICLE IV, SECTION 1: "The planning of the work of the Association; arrangements for meetings and programs and any or all other matters pertaining to the administration of the affairs of the Association shall originate in the House of Delegates but all its actions must be ratified by the Board of Directors."

ARTICLE IV, SECTION 2: "The Board of Directors shall have charge of the property and financial affairs of the Association and shall perform such duties as are prescribed by the Constitution and By-Laws and the State law governing directors of corporations. It must ratify the transactions of the House of Delegates, Committees and Boards before they can become effective."

All sections to be renumbered: Section 1 becoming Section 3, etc.

ARTICLE IV, SECTION 4: "The Managing Director shall be the executive officer of the Executive Committee and, under its supervision, during the intervals between meetings, shall manage, direct and conduct the affairs of the Association. He shall supervise and direct the activities of the Executive Secretary and shall render a report on the status of the Association to the Board of Directors at their Annual Meeting."

ARTICLE IV, SECTION 2 and 3: Delete the word "Secretary" in the compound word "Secretary-Treasurer" and the last one in section 3 should be changed to "Managing Director."

Trauma and Glomus Tumors

—*Relationship, with Report of a Case*—

MARTIN J. LOEB, M.D.,
New York City

WISEMAN in 1676 considered the bruise among other things as the causation of Cancer.[1]

Ewing states that mechanical trauma is an important factor in the causation of tumors.[2]

In his later monograph[3] Ewing reviews all the data relating to traumatism as the causation of tumors both benign and malignant. He arrives at the following conclusion: "Present data confirms the view long since adopted by pathologists that a single trauma of normal tissues is incapable of producing a malignant tumor. This principle may not greatly alter the possible importance of a single trauma as an indirect but an essential and determining cause of certain tumors." It is interesting to note that in this report Ewing omits glomus tumors.

Geschickter[4] tabulates 78 cases of glomus tumors which he collected up to 1936 giving incidents in various parts of the body but in his tabulation he features mainly clinical phenomena and particularly omits the causation.

From the Service of DR. LOEB, Bronx Hospital.

Mason and Weil[5] in a review of their cases, considered trauma to be responsible for the tumor in 36%.

Grauer and Burt[6] state that in 40 to 50% of glomus tumors there was a previous history of trauma. The first cases reported are those by Barré in 1922. The very first case however, Barré reported in 1920 to the Congress of Neurology at Strasburg.[7] It is interesting to note that the first case ever to be reported (Barré's) was that of a girl of 13 years of age who developed a glomus tumor subsequent to an injury. The history of the first case is as follows:

A cover of a school desk fell on the middle left finger of this 13 year old girl. A painful hematoma appeared under the nail which lasted five weeks after which the hematoma disappeared and the pain subsided. After a few weeks however, severe pain began to manifest itself again, first in the finger and finally involving the entire extremity. Temperature of the affected side was elevated in comparison to the one of the opposite side. There was slight atrophy of the affected side. Symptoms lasted five years. Resection of the lateral nerves gave no relief. The nail was then incised and small tumor removed after which the girl was rapidly cured.

The third case of Barré occurred on the plantar surface of the foot at the center of the metatarsal arch which naturally is subject to trauma particularly if the arch be for some reason disorganized. Barré reported these cases but did not make a thorough study of his material.

From the medicolegal point of view, the question of traumatism is of paramount importance, particularly in compensation cases. Whether the tumor is compensatory or not depends upon traumatism and the causal relationship between the traumatism and the injury received. At the end of this paper, a table of 144 cases is appended wherein the relationship of the tumor to injury is tabulated. Since the tumor may occur long after the injury has been sustained, it stands to reason that the patient may forget that he sustained an injury at the site of the tumor and the physician may forget to inquire in order to establish the relationship. It is quite possible that this table does not adequately represent the actual percentage of glomus tumors caused by injury. The percentage is probably much higher.

Mason[8] studied the material of Barré's cases as well as a glomus tumor which he observed in 1916. His description was the first study made of the tumors of the glomus.

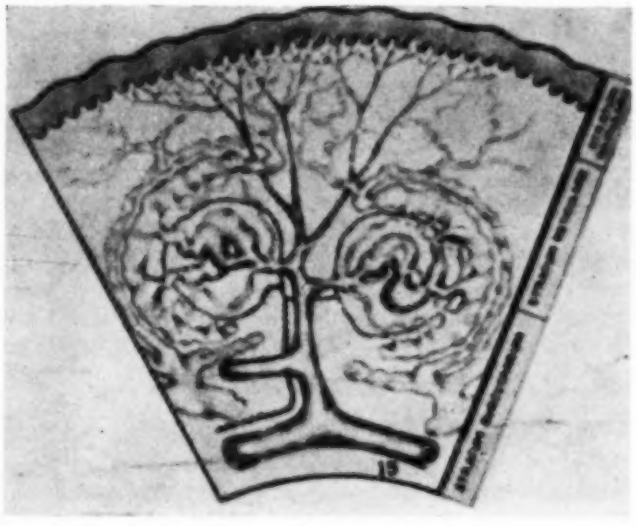
Popoff's[9] investigation of the glomus following those of Masson have contributed greatly to the conception and understanding of the subject.

Definition

ONE CAN define the glomus as a thermo regulatory organ located in any part of the skin particularly in the hands and feet and under the nails where heat regulation is more necessary. It acts both as a regulator of the local as well as that of the systemic temperature.

The normal glomus is a shunt between the arterial system and capillary arterial system from which efferent veins emerge.[10] The glomus is a neuro-muscular-arterial organ and is controlled by the sympathetic nervous system. Subject as it is to nervous influences the dilatation and contraction of the arterial system in the glomus is controlled by the nervous mechanism of the glomus. Popoff credits Hoyer for the anatomic studies of the arterial venous anastomoses and gives Hoyer credit as being the father of the anatomic conception of the glomus. Sucquet preceded Hoyer with the description of ordinary capillaries which open directly into the neighborhood vein without forming a venous capillary network.

Popoff's conception of the glomus is hereby appended as well as the description of his diagram:



Diagrammatic presentation of vascular arrangement and the glomus, as found in the ventral surface of the digit. It shows: (1) all the zones of the skin, including that occupied by the glomic apparatus; (2) the afferent artery of the glomus; (3) the coiled type of Sucquet-Hoyer canal, characterized by a thick wall; (4) the efferent part of the Sucquet-Hoyer canal entering the primary collecting vein, with the latter appearing as a long, wide ruffle encircling the glomus; (5) the relation of the primary collecting vein to other veins; (6) the system of preglomic arterioles supplying all the constituents of the glomus and emptying into the primary collecting vein, and (7) division of the periarterial nerve trunks, with branches going to the glomus. This diagram serves to explain arteriovenous and trophic disturbances caused by functional disability and organic destruction of either the entire glomus or one of its constituents.

Location

THE tumor is usually found in the extremities particularly under the nails although it may be found anywhere in the body. Grauer and Burt[6] report two cases which they found in the male genitals. The tumors vary in size from five to 10 millimeters and may reach up to two centimeters in diameter. They may be purple or rose colored. The glomus tumor may be regarded as an exaggerated normal structure instead of a new growth.

Description

THE description of the tumor has been given by Stout[11] as follows:

The glomus tumor on cross section is seen to consist of a small angled mass of blood-vessels enclosed within a capsule. Microscopically, the vessels are endothelial-lined and supported by a fine fibrous network. The rest of the wall is made up of peculiar cuboid or rounded "glomus" cells (usually referred to as epithelioid cells of Masson) and smooth muscle well differentiated or in an embryonal form in which the smooth muscle fibers are found within the cytoplasm of the epithelioid cells. The glomus cells are quite distinctive, having well defined cell outlines, accentuated by delicate collagen fibers which separate every cell from its neighbor. The cytoplasm is pale, sometimes vacuolated, which brings into sharp relief the nucleus which is voluminous, centrally placed, globular or ovoid. Myelinated nerves are generally found in bundles in or near the capsule (can be seen in ordinary stains). Also present are numerious slender non-myelinated nerve fibers beneath the capsule and the epithelioid cells.

Symptomology

THE most important symptom in the glomus tumor is pain, pain which is constant, exquisite and agonizing in character, burning at times because of the nerve involvement. The pain may be present long before the tumor is discernible. The pain may come in paroxysms lasting from several minutes to several hours. It may be localized to the area of the tumor or may radiate upward to the shoulder or the neck. Pain is also caused by the slightest touch such as pressure of clothes or change of temperature, particularly to cold. A paroxysm of pain brought about by this means may last for several hours. Pain and the appearance of the tumor, one a subjective symptom, the other an objective sign, are the best aids to the diagnosis.

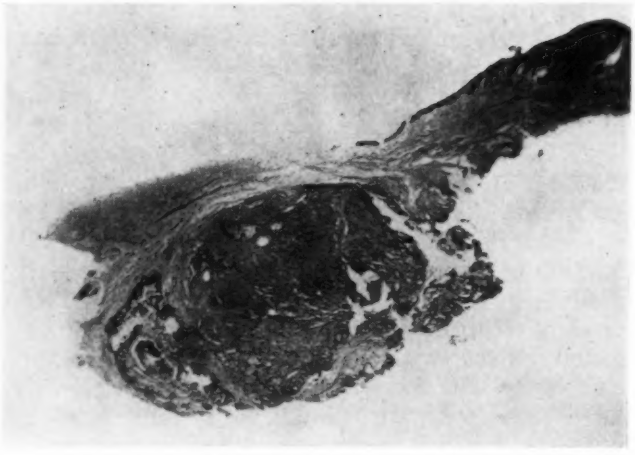
History of the Case

MACHINIST. S. S. 31 years. Referred on June 23, 1937, with the following history:

Eight years ago the patient's left ring finger was caught in a machine and squeezed between two plates of that machine. There was a discoloration of the finger which lasted for several weeks. Since that time he has had pain in that finger particularly on the slightest touch. The pain was more severe during the winter time or on sudden change of temperature. At times he would get paroxysms of pain which would shoot up to the shoulder. These pains, which were very severe, lasted from one to several minutes at a time.

PHYSICAL EXAMINATION: Patient presented a small purple spot under the finger about the size of the head of a pin. He experienced exquisite pain on the slightest touch. He was admitted to the hospital on the second day of July, 1937. Under a local infiltrating anaesthesia the nail was split open, part of nail removed and the small tumor was excised. He was seen again on October 7, 1939. He claims that he occasionally has a slight pain going up to the shoulder but it is not sufficient to disturb him. His finger is not sensitive to touch and is able to work with it, although he favors it on occasions.

The illustrations of the tumor which we have removed were described by Dr. J. Felsen, Pathologist of our Hospital.



Glomus tumor is surrounded by a thick connective tissue capsule which appears to divide it near one end

Pathology

GROSS: Specimen consists of an 0.5 c.m. piece of firm white tissue.

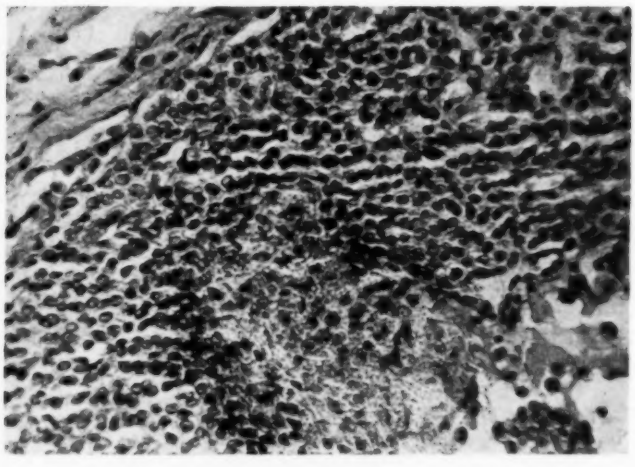
MICROSCOPIC: Section shows a well encapsulated cellular tumor which lies deep in the subcutaneous tissue. The overlying subcutis contains many small blood vessels. The tumor proper seems to be divided by a fibrous septum which is continuous with the thick connective tissue capsule. The tumor contains many thin-walled blood vessels about which are arranged typical glomus cells that bear a close resemblance to epithelial cells. The nuclei are large, round, somewhat vesicular and contain small, centrally placed nuceoli. The cytoplasm is acidophilic. Here and there some smooth muscle fibers can be discerned, particularly in the neighborhood of the blood vessels. The characteristic "S" shaped or coiled arteriovenous anastomosis is not seen, but one or two large thin-walled cavernous-like blood spaces are present. There is a rather sparse collagenous reticulum in which a few nerve fibers can be seen.

DIAGNOSIS: Arterial angioneuromyoma of Masson.

Lower power photomicrograph showing location of glomus tumor in subcutaneous tissue. Note numerous small blood vessels both in the tumor and in the intervening tissue between it and the epidermis

Illustration

The writer was interested to obtain definite data on the relationship of trauma to glomus tumor. Authors varied greatly in their statistics. Some attribute the causation to injury in a larger percentage of cases than others do. The analysis of 144 cases is hereby made of which 31 are definitely reported as having an antecedent history of trauma. Four cases are questionable because of the length of time or because of an indefinite history. This gives a percentage of 20.85% caused by injury.



High power photomicrograph showing the characteristic glomus cells with large round nuclei and nucleoli, which bears a strong resemblance to epithelial cells

Summary and Conclusion

A CASE of glomus tumor is presented with a definite history of traumatism. The tumor occurred shortly after, and the patient suffered for eight years before he was cured by operation.

2. The normal glomus as well as the pathology of the glomus tumor is reviewed.

3. An analysis of 144 cases is hereby appended emphasizing the relationship of traumatism to the causation of the tumor.

GLOMUS TUMORS
1920-1939
SHOWING NUMBER CAUSED BY TRAUMA

Author	Date	Total Cases	History of Trauma	Time Elapsed	Remarks
Adair	1934	10	0		
Bailey	1935	7	1	20 y.	
Barre	1920	1	1	8-10 weeks	
Barre	1922	3	0	..	4 reported, but 1 same as above
Barre and Masson	1924				2 reported, but same as above
Bergstrand	1937	2	1	2 yrs.	
Blanc	1924	1	0		Later reported as Case 2 by Masson and Gery
Blinder	1939	1	1	15-20 y.	
Bonnard et al	1934	2	0		
Bonnet	1927	1	0		
Burman and Gold	1935	2	0		"Subungual angiosarcoma"
Carstensen	1927	1	0		
Cole and Stroub	1936	1	0		
Cooper	1930	1	1	4 m.	
Costa	1932	1	0		
Doane	1939	1	1	21 y.	Contusion of **ring finger**; tumor on **middle finger.**
Dupont	1931	3	0		No clinical histories.
Fernandez and Monserrat	1931	1	0	4 y.	Both **ears.** Histologically verified.
Fischer-Wasels	1931	2	0		
Freudenthal, et cl	1937	1	0		
Gay Prieto	1935	2	0		
Genner	1930	1	0		
Gerundo	1938	1	0		
Greig	1928	3	1	10 y.	
Hagieteanu	1934	2	1		Present age 34. As child injured leg.
Hopf	1930	4	1 (?)	48 y.	5 reported, but 1 was **Wegelin's** (q.v.)
Horsley	1936	1	1	15 y.	
Ianichewski and Lebel	1928	1	0		
Kendall and Thomson	1938	3	1	10 y.	
Geschickter	1936	2	0		2 new cases; 17 already reported by Lewis and Gesc.
Jirka and Scuderi	1936	1	1	10 y.	
Kline and Brody	1939	2	0		
Kolodny	1938	1	0		
Lewis and Geschickter	1935	17	2	a. "many years" b. 6 y.	
Loeb, M. J.	1937	1	1	8 y.	
Lortat-Jacob and Brosse	1928	1	0		
Love	1935	1	0		
Mackey and Lendrum	1936	3	1	7 y.	
	1939	13	3	10 y. 12 y. 20 y.	
Martin and Dechaume	1925	2	2	a. ? b. 25 y.	
Mason and Weil	1934	1	1	37 y.	
Masson	1924	1	1	5-6 weeks	3 cases reported, but 2 already rep. by Barre (q.v.)
Masson	1927	1	0		
Masson and Gery	1927	3	1	15 y.	4 reported, but 1 already rep. by **Blanc** (q.v.)
Meyers	1939	4	0		
Mueller, S. C.	1939	1	0		Subungual glomus tumor
Nicod	1927	1	0		
Paulian et al	1933	1	0		
Picard	1931	1	0		
Prodanoff	1927	1	1 (?)	22 y.	Fall, injuring head and left hand. Tumor on left **thigh.**
Radasch	1937	4	0		
Raisman and Mayer	1935	3	1	12 y.	
Schoch and Aronson	1936	1	0		
Saccone and Mendeloff	1939	1	1		
Stout	1935	11	2	8 y.	The questioned case was a supposed injury in infancy.
			1 (?)	18 m.	
Stratmann	1933	1	0		
Theis	1937	1	0		
Thomas	1933	1	1	2 y.	
Wegelin	1927	1	0		"Arterielles Angiomyoneurom"
Weideman and Wise	1937	1	0		5 other cases mentioned in discussion.
		144	31		
Some Earlier Cases (probably glomus)					
Chandelux	1882	1	0		"Nevrome corpusculaire sousungueal douloureux."
Kolaczek	1878	1	0		"Angio-Sarkom"
Kraske	1880	1	1		"Subunguales Sarkom"
Kraske	1882	1	1		Injury in childhood.
Muller	1901	1	1	23 y.	"Subunguales Peritheliom"
Wood	1812	8	1	10 y.	"Painful subcutaneous tubercle"

References:

1. Quoted by BEHAN in "Relation of Trauma to New Growths" Williams & Wilkins Co., Baltimore, 1939.

2. EWING, JAMES: Neoplastic Diseases, 3rd Edition. W. B. Saunders, 1928, page 118.

3. EWING, JAMES: *Arch. Path., Vol.* 19, 701, 1935.

4. GESCHICKTER, CHAS. F.: *Internat. Clin.*, Philadelphia. Vol. 2, 1936, p. 1.

5. MASON, M. L., and WEIL, ARTHUR: Tumor of Surg. gynec. obst. Vol. 58, pages 807-816, May, 1934.

6. GRAUER, ROBT. C., and BURT, JAMES C.: *J.A.M.A.* Vol. 112, No. 18, May 6, 1939, p. 1806.

7. BARRE, A.: Sur certaines Sympathalgies de la peripherie des membres. Leur traitment—chirurgical Simple. *Paris Med.*, October, 1922. Also quoted by P. MASSON: "Le glomus Neuro-myo-arteriel des regions tactiles et ses, tumeurs" Lyon Chirurgical tome XXI, p. 17, May-June, 1924.

8. MASSON, P. Same as above.

9. POPOFF, NICHOLAS W.: *Arch. Path.*, Vol. 18, No. 3, p. 295, September, 1934.

10. HUMPHREY, ARTHUR A., and BRAINARD, CLIFFORD W.: *Int. Surg. Digest*, Vol. 24, No. 5, page 259, November, 1937.

11. STOUT, A. P.: *Am. J. of Cancer*, Vol. 24, p. 255, June, 1935.

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ALVAREZ, CASCOS, M., and COSTERO, I.: *Arch. españ. de oncol.* 2: 391, 1932. Cited by Mason and Weil. (Not in NYAM library.)

BAILEY, O. T.: The cutaneous glomus and its tumors—glomangiomas. *Am. J. Path.* 11:915, 1935. Reports seven cases, one traumatic (see table). *Case —M. 48. Left upper arm. Followed blow, 20 years ago.

BARRE, J. A.: Troubles sympathiques étendus et violents du membre superieur par tumeur du doigt. *Rev. neurol.* 27:942, 1920. *F. ?. Left middle finger. Slight injury in 1914.

BARRE, J A.: Sur certaines sympathalgies de la périphérie des membres . . . *Paris méd.* 12:pt.2, 311, 1922. Four cases. Case 1 already reported (see previous entry). Case 2. M.?. Left index finger. 30 y. Case 3. M.35 Sole of foot. 8 m. Case 4. F.50. Over lower end of radius. 6 m.

BARRE, J. A, and MASSON, P.: Etude anatomo-clinique de certaines tumeurs sous-unguéales douloureuses. *Bull. Soc. franc. de dermat. et syph.*, 31: Réun. dermat. de Strasbourg, 148, 1924 Case 1. F.18. Left middle finger. Desk lid fell on nail 5 y. earlier (this is probably Barré's first case.) Case 2. M.? Left index finger. 30 y. (This is probably Barré's second case.)

BERGSTRAND, H.: Multiple glomic tumors. *Am. J. Cancer*, 29:470, 1937. Case 1. M.33. Pain in ankle at 8-9 y. *Case 2 M.21. Left foot. Symptoms appeared first in February, 1935, after fall; foot became swollen and tender May, 1936, after he slipped.

BLANC: Neurogliome sous- custané douloureux chez un veillard de 82. *Loire méd.*, 38:374, 1924. M.82. Right thigh. 12 y. ((This case is one later reported by Masson and Géry.)

BLINDER, S.: Glomus tumor; report of a case. *Am. Heart J.*, 17:238, 1939. M.38. Nail of left index finger. 15-20 y

BONNARD; DE GRAILLEY, R., and BONNARD, H.: Les tumeurs glomiques. *Gaz. hebd. d. sc. méd. de Bordeaux*, 55:498, 1934. Case 1. M.35. Middle finger. 2 m. Case 2. M.56. Right middle finger. 4 y.

BONNET, P.: Tumeur sous-unguéale douloureuse. Tumeur du glomus neuromy-artériel. *Lyon chir.* 24:718, 1927. F.46. Right little finger. Since childhood.

BURMAN, O. S., and GOLD, A. M.: Glomus tumor. *New York State J. Med.* 35:618, 1935. Case 1. M.62. Right knee. Since 1919. No definite history of trauma. Case 2. F.45. Left knee. 1 m.

CARSTENSEN, I.: Ueber subunguale Tumoren. *Arch. f. klin. Chir.*, 144: 409,1927. (Reviews earlier literature—"subunguales Angiosarkom"). Case (Roedelius). F.27. Right forefinger and ring finger. 12 y.

COLE, H. N., and STROUB, W. E.: Glomus tumor; arterial angioneuromyoma of Masson. *J.A.M.A.* 107:428, 1936. F.33. Left little finger. 77 y.

COOPER, T. C.: A case of "perithelioma" of facial origin with a direct antecedent history of trauma. *Lancet*, 2:1234, 1930. *F.38. Elbow. 4 m. Blow on elbow. Cited by STRATMANN. Rejected by STOUT.

COSTA, A. J.: Tumor del glomus neuro-mio-arterial del dedo indice izquierdo. *Bol. y trab. Soc. de cir. de Buenos Aires*, 16:1514, 1932. F.50. Left index finger. 10 y.

DOANE, C. P.: Glomus tumor (glomangioma). *J.A.M.A.* 112:1049, 1939. F.31. Left middle finger. Contusion of ring finger when she was 10, but middle finger was not involved in accident.

DUPONT, A.: Aspects atypiques des tumeurs glomiques. *Rev. beldge d. sc. méd.*, 3:624, 1931. Three cases, with no clinical histories. Case 2. Finger. Case 3. F.41. Left fourth finger. 16 y.

Note sur quelques aspects atypiques des tumeurs glomiques. *Bull. Soc. franc. de dermat. et syph.*, 42:1260, 1935. Histology. No detailed case reports.

FACIO, L.: Tumor subungual doloroso. *Rev. argent. de dermat. y sif.* 13:332, 1929. Cited by STOUT. (Not in NYAM library.)

FERNANDEZ, A. A., and MONSERRAT, J. L. E.: Nódulas doloros de la oreja (tumor glomio; neuro-mio-angioma). *Semana méd.* 38:pt.2, 1993, 1931. M.38. Helixes of both ears. 4 y. Questioned by RAISMAN and MAYER, but verified histologically

FISCHER-WASELS, B.: Zwei Angioneuromyome . . . *Centralbl. f. Path.* 50:388, 1931. *(Versamml westdeutsch. Path. u. Aerzte).* Case 1. M.35. forearm. Case 2. M.81. thigh.

FREUDENTHAL, W., ANDERSON, R. G., and WEBER, F. P.: The glomus and the glomus tumour (Masson), with the clinical account of a case. *Brit. J. Dermat.*, 49:151, 1937. F.50. Left little finger. 25 y.

GAY PRIETO, J.: Contribution á l'étude des terminaisons nerveuses dans les tumeurs glomiques de P. Masson. *Bull. Soc. franc. de dermat. et syph.*, 42:1254, 1935. Case 1. F.43. Left arm. Some months. Case 2. F.92 Left calf. Some months.

GENNER: Tumeurs du glomus neuro-myo-artériel des extrémites. *Zentralbl. f. Haut—u. Geschlechtskr.*, 33:27, 1930 *(Danish dermat. Soc.)* F.33. Right index finger. 13 y.

GERUNDO, M.: Glomic tumor with report of a case. *J. Kansas M. Soc.*, 39:506, 1938. F.36. Left little finger. 8 y.

GREIG, D. M.: Subcutaneous glomal tumours: painful subcutaneous nodules. *Edinburgh M. J.* 35:565, 1928. Case 1. M.70. Right thigh. Many years. *Case 2. M.42. Right deltoid region. 10 y. Fell on right forearm. Case 3. F.33. Left calf. 8 y. Fractured left leg 6 y. before present time, but swelling was already present.

HAGIETEANU, M: Contributiuni la studiul tumorilor glomici nevralgice. *Spitalul*, 54:114, 1934. *Case 1. F. 34. Right lower leg. As child injured right leg. Noticed pains 4 y. ago. Case 2. F.21. Right lower leg. 2 y.

HOPF, M: Uber Tumoren des neuromyoarteriellen Glomus (Masson). *Frankfort. Ztschr. f. Path.*, 40:387, 1930. Case 1. M.50. Left acromion. 10 y. Case 2. F.78. Palm. Several years. Case 3. See *Wegelin.* Case 4. M.65. Thigh. 4 y. ? Case 5. M.65. Calf. 48 y. Kicked in leg (not proven).

HORSLEY, G. W.: Glomus tumor; case report. *South. Med. & Surg.*, 98:5, 1936. *F.27. Palm of right hand. 15 y. Bruise from can opener.

IANICHESWKI, A., and LEBEL, M.: Une variété de nevralgie; la sympathalgie due a une tumeur glomique. *Presse méd.*, 36:116, 1928. F.50. Left ring finger. 18 y.

KENDALL, A. W., and THOMSON, S.: Glomus tumors. *Lancet*, 1:1102, 1938. *Case 1. M.38. Left knee. 10 y. Knocked knee while getting off bus; 5 y. later small nodule. Case 2. F.42. Right thumb. 12 y. Case 3. F.51. Right thumb. 5 y.

KLINE, P. R., BRODY, M., and BRODY, E. R.: The glomus tumor; report of two cases. *Ohio State M. J.*, 35:1317-1319, December, 1939. Case 1. M., 42. Left arm. 8 years. Case 2. F., 27. Left middle finger nail. 6 years.

GESCHICKTER, C F.: Glomal tumors. *Internat. Clin.*, 46 s. 2:1, 1936. Cases 1-17 previously reported (see Lewis & Geschickter). Case 18. M.40. Wrist. 20 y. Case 19. M.56. Knee. 37 y.

JIRKA, F. J., and SCUDERI, C. S.: Glomus tumor; report of a case. *J.A.M.A.*, 107:201, 1936. *M.70. Arm. 10 y. Fall.

KOLODNY, A.: Glomus tumor (glomangioma). *Ann. Surg.*, 107:128, 1938. F.22. Right middle finger. 10 y.

LEWIS, D, and GESCHICKTER, C. F.: Glomus tumors. *J.A.M.A.*, 105:775, 1935. Case 1. F.38. Right little finger. 8 y. *Case 2. M.67. Left knee. Fracture many years before. Case 3. F.67. Right elbow. 15 y. Case 4. F.38. Right thumb. 3 y. Case 5. M.65. Forearm. Case 6. M.47. Right thigh. Case 7. F.30. Thigh. 3 m. Case 8. M:45. Right leg. Many years. Case 9. M.57. Knee. Case 10. F.27. Palm of hand. Case 11. M.48. Left arm. Case 12. F.60. Finger. 15 y. *Case 13. M.29. Left elbow. 6 y. Fall. Case 14. M? Left wrist. 8 y. Case 15. M.80. Dorsal surface of hand. Case 16. M.51. Right elbow. 12 y. Case 17. F.24. Right thumb. 1 m.

LORTAT-JACOB, L., and BROSSE, T.: Tumeur sous-unguéale violacée et douloureuse avec causalgie du membre supérieur (glomus tumoral neuro-myo-artériel). *Bull. Soc. franc. de dermat. et syph.*, 35: 305, 1928. F.41. Left ring finger. 16 y.

LOUTCHITCH, M.: Tumeurs sous-unguéales douloureuses (angio-myo-neuromes artériels); tumeurs glomiques. *Lyon thesis*, 1927. Summarizes 14 cases, none original.

LOVE, J. G.: Tumor of a subcutaneous glomus . . . report of a case. *Proc. Staff Meet. Mayo Clinic*, 10:593, 1935 M.38. Left elbow. 2 y.

MACKEY, W. A., and LENDRUM, A. C.: Three cases of glomangioma or angio-neuromyoma (painful subcutaneous tubercle). *Brit. J. Surg.* 24:208, 1936. Case 1. M.24. Left shin. 14 y. Case 2. M.59. Elbow. *Case 3. M.12. Knee. 7 y. Blow with cricket ball. Glomangiona: a form of "painful subcutaneous tubercle." *Brit. M. J.*, 2:676, September 30, 1939, 13 cases (total 15, but two already reported).

MUELLER, S. C.: Subungual glomus tumor. *Minnesota Med.*, 22:708, October, 1939. F.66. Left ring finger 17. No history of trauma.

MARTIN, J. F., and DECHAUME, J: Les tumeurs glomiques (angio-neuromyomes artériels). *Ann. d'anat. path.*, 2:239, 1925. *Case 1. F., ca. 40. Finger. Crushing injury of first joint. *Case 2. F.70. Right ring finger. 25 y. Slight injury.

MASON, M. L., and WEIL, A.: Tumor of a subcutaneous glomus. *Surg. Gynec. & Obst.*, 58:807, 1934. *M.56. Left knee. 37 y. Bicycle accident, knee struck steel rail.

MASSON, P: Le glomus neuromyo-artériel des régions tactiles et ses tumeurs. *Lyon chir.*, 21:257, 1924. Case 1. (seen in 1916). F.23. Forefinger. 5-6 weeks. Case 2. Reported by Barré, 1920. (q.v.) Case 3. Reported by Barré, 1920. (q.v.)

Etube sur le glomus. *Arch. per l. sc. med.* 50:47, 1927 Says he has seen seven cases Reports one: F.35. Left thumb.

Les glomus cutanés de l'homme. *Bull. Soc. franç. de dermat. et syph.*, 42:1174, 1935. Tumors, p.1203. In 18 years he has seen 27. No case reports.

MASSON, P., and GERY, L.: Les tumeurs glomiques sous-cutanés en dehors des doigts. *Ann. d'anat. path.*, 4:153, 1927. Case 1. M.69. Forearm. Case 2. Reported by BLANC, 1924 (q.v.) *Case 3. M., "middle-aged." Thigh. Injury 15 y. before. Case 4. ? ? Arm (no details).

MEYERS, M. P.: Glomus tumor; report of four cases. *J. Michigan M. Soc.*, 38:204, 1939. Case 1. F.54. Right lower thigh. 3 y. Case 2. F.37. Right middle finger. 5 y. Case 3. F.27. Right fourth finger. 10 y. Case 4. M.35. Right fourth finger. 10 y.

NICOD, J. L.: Le glomus neuromyo-artériel sous-cutané et ses tumeurs. *Schweiz. med. Wchnschr.*, 57:1177, 1927. F.31. Left thumb. 4 y.

PAULIAN, D.: Stefan-Popescu, and Marinesco-Slatina, D.: Tumeur glomique sous-unguéale suivie de'hémihypertrophie et guérison complète après l'ablation chirurgicale. *Ann. d'anat. path.*, 10:271, 1933. F.32. Right middle finger. "A long time"

PICARD, H: Uber seltene Tumoren am Nagelbett (Neuromyoarterielle Glomustumoren). *Zentralbl. f. Chir.*, 58:2133, 1931. M.49. Left forefinger. 1½ y.

POPOFF, N. W.: The digital vascular system. *Arch. Path.*, 18:295, 1934. General; no tumors.

SACCONE, A., and MENDELOFF, J.: Glomus Tumor of arm. *J. Lab. & Clin. Med.*, 25:245-250, December, 1939. M., 42. Left arm. Painless nodule ("blue birthmark") on arm for 10 years. Trauma eight months before admission to hospital since nodule has been painful.

PRODANOFF, A.: Sur la localisation des tumeurs glomiques (angio-neuromyome de P. Masson). *Ann. d'anat. path.*, 4:147, 1927. ?M.68. Left *thigh.* 22 y. Fall, injuring head and left *hand.*

RADASCH, H. C.: Glomal tumors. Arch. Path., 23:615, 1937. Case 1. F.26. Left thumb. 10 y. Case 2. F.30. Middle finger. 5 y. Case 3. F.35. Left arm. 4 y. Case 4. F.22. Thumb. 5 y.

RAISMAN, V, and MAYER. L.: Tumor of the neuromyo-arterial glomus; report of cases. *Arch. Surg.*, 30:911, 1935. *Case 1. F.28. Right third finger. 12 y. Drawer slammed on finger. Case 2. F.47. Left little finger. 18 M. Case 3.F. 35. Right ring finger. ca. 2 y.

SCHOCH, A. G., and ARONSON, H. S.: Glomus tumor. *Texas State J. Med.*, 32:545, 1936. F.31. Right thumb. 8 y.

STOUT, A. P.: Tumors of the neuromyo-arterial glomus. *Am. J. Cancer*, 24:255, 1935. ? Case 1. F.31. Left middle finger. 2 y. Injury in infancy (?) Case 2. F.38. Left little finger. 13 y. Case 3. F.43. Right ring finger. 4 weeks. *Case 4. F.28. Finger. 8 y. Severe crushing injury. Case 5. F.30. Left little finger. 8 y. *Case 6. M.59. Forearm. 18 m. Blow with stick. Case 7. M.56. Left forearm. 1 y. Case 8.

M.65. Right forearm. 43 y. Case 9. M.59. Left gluteal region. 2 y. Case 10. M.?. Leg. No history. Case 11. F.25. Heel. "Several years."

STRATMANN, A.: Uber seltene Tumoren im Nagelbett. *Dermat. Ztschr.*, 67:129, 1933. F.21. Left fourth finger. 3 y.

THEIS, F. V.: Subungual neuromyo-arterial glomus tumor of the toe. *Arch. Surg.*, 34:1, 1937. M.69. Right first toe. More than a year.

THOMAS, A: Tumeurs comparables a des tumeurs glomiques développées dans les muscles de la cuisse a la suite d'un traumatisme. *Ann. d'anat. path.*, 10:667, 1933. *M.27. Left thigh. 2 y. Fall from bicycle, handlebars striking knee and inside of leg.

WEGELIN, C.: Arterielles Angiomyoneurom. *Schweiz. med. Wchnschr.*, 57:895, 1927. M.47. Calf. "Several years."

WEIDMAN, F. D., and WISE, F.: Multiple glomus tumors of the order of telangiectases. *Arch. Dermat. & Syph.*, 35:414, 1937. F.35. Multiple (48) painless lesions on extremities, cheeks, shoulders, buttocks. 20 y. (?) Other cases mentioned in discussion.

CASES BEFORE 1920

(Often cited, but questioned by some authors as not proved)

CHANDELUX, A.: Recherches histologiques sur les tubercules sous-cutanés douloureux. *Arch. de physiol. norm. et path.*, 9:639, 1882. Review of early literature; also: Case C. "Névrome corpusculaire sous-ungual douloureux." F.16. Left right finger. 2 y.

KOLAKZEK, J.: Ueber das Angio-Sarkom. *Deutsche Ztschr. f. Chir.*, 9:165, 1878. Case 2. F.42. Right great toe. 4 y.

KRASKE, P.: Subunguales Sarkom des linken Mittelfingers. *Centralbl. f. Chir.*, 7:609-612, 1880. F.42. Left middle finger. 12 y.

Ueber subunguale Geschwülste. *München. med. Wchnschr.*, 34:889, 1887. *F.40. Left thumb. Injury in childhood

MULLER, R F.: Zur Kenntsis der Fingergeschwülste. *Arch. f. klin. Chir.*, 63:348, 1901. *Case 16. "Subunguales Peritheliom." F.44. Left fourth finger. Injured while spinning 23 years before.

VINCENT, J.: Des tumeurs sous-unguéales douloureuses. *Lyon thesis*, 1900. (Not in NYAM library.)

WOOD, WILLIAM: On painful subcutaneous tubercle. *Edinburgh M. J.*, 8:283, 1812. Reports eight cases, one traumatic: *Case 7. F.57. Right calf. 10 y. Rupture of fibers of gastrocnemus muscle.

Work Output and Pregnancy

NORMAN J. POWELL,
Director of Research,
New York City Civil Service Commission

CONSIDERABLE variation exists both in the public service and in private industry in respect to personnel policies adopted for dealing with pregnant females. The problems are, first, to establish the maximum period during which females will be permitted to work during their pregnancy and, second, to fix the minimum period following childbirth after which the mother will be permitted to return to work.

The factors determining the nature of the policy to be established are the extent to which the physical appearance of the pregnant female is relevant to the proper execution of her duties and the relationship between work efficiency and the condition of pregnancy or of status subsequent to childbirth.

It is the purpose of this investigation to ascertain the relation between work output and pregnancy. Two females have been studied, one serving as the experimental, the other as the control subject. The women are quite closely matched in regard to the major variables. Both are in the late twenties, white, American, in excellent health, and approximately equally experienced as clerical employees. This was a first pregnancy for the experimental subject. Both were engaged in a simple clerical task in which only slight physical effort was required. The job was that of rating examination papers with the use of a stencil. Test answers were in the form of letters, A, B, C, or D. Scoring was done by counting the number of correct letter designations for the answer.

Data have been computed in terms of number of papers rated per hour and grouped in intervals of one week.

The trend in speed of work output is similar for both women. In general, both show a tendency to improve and both fluctuate in about the same way.

In order to validate the possibility that pregnancy is an important factor in influencing clerical efficiency at this simple task, there should be a steadily increasing gap between the output of the subjects. This is distinctly not the case. The control appears to be a superior worker in comparison to the experimental employee, and the variable conditioning the difference in quality is not pregnancy. The median difference in rates during April is eight per hour; in July the difference is only three. Upon the experimental subject's return to her employment, she worked at a rate closely in line with her previous speed.

NUMBER OF PAPERS MARKED PER HOUR BY PREGNANT AND CONTROL SUBJECT

Date	Rate Of Pregnant Subject	Rate Of Control Subject	Control Minus Pregnant Subject's Rate
April 1	19	28	9
8	11	24	13
15	22	24	2
22	24	25	1
29	20	28	8
May 6	23	28	5
13	25	27	2
20	24	25	1
27	22	34	12
June 3	20	30	10
10	21	26	5
17	23	26	3
24	22	29	7
July 1	26	29	3
8	29	30	1
15	26	21	–5
Oct. 21	21		
28	28		

Quantitative data have been available only from April 1 through July 19, and from October 21 through October 31. The experimental subject gave birth July 28, 1940. It is, of course, possible that during the experimental subject's period of pregnancy prior to April 1, her work output was subnormal. Since, however, the data analyzed show that pregnancy has no effect on the quantity of work done for the four months immediately preceding the subject's delivery, it seems reasonable to believe that in all probability is not related to efficiency even during the early pregnancy period. The assumption is that pregnancy would exercise an increasingly vigorous influence as parturition is approached.

The finding then, is that in the situation studied, no relationship exists between pregnancy and efficiency. While this is an inadmissible generalization because of the limitations of size of experimental population and the character of the job studied, another generalization may be made. The condition of pregnancy does not necessarily result in a decrease in amount of work done in performing a simple clerical function. This investigation does not support the reasonableness of enunciating a general policy which will cover all cases of pregnant females.

At least the reasonableness of any general policy must be rooted in considerations other than efficiency correlatives. It may be held that females

in the late stages of pregnancy are unsightly or unbusinesslike in appearance. Or it may be believed that it is administratively easier to deal with pregnant employees as a class, not as individuals.

But it cannot rightly be stated that a female necessarily becomes incompetent temporarily while she is pregnant.

The Western Association

—First Annual Meeting—

THE WESTERN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS, whose charter meeting is being held at Del Monte, on Sunday, May 4, 1941, has been a component society of the AMERICAN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS since January 1, 1941. Its objectives are to promote interest and education in industrial medicine and in its problems, which include:

Physical examination of employees.

Plant sanitation and preventive medicine in industry.

The problem of placement of partially disabled or aged employees.

The medical or surgical care of those disabled through occupational accidents or disease.

The rehabilitation of employees not adequately adjusted following injuries or disease, as well as

The status, interpretation and trends of compensation laws.

There are two classes of memberships: (A) Active membership — which includes those members who are engaged at least 25% of their time in industrial medicine and are responsible for the medical environment of the employees of a unit of industry; they must belong to their respective county medical societies and be either members or Fellows of the American Medical Association; these members automatically become members of the AMERICAN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS; and (B) Associate membership, which comprises those members who, through their specialties and practice, treat industrial injuries or disease, but whose connection with the industrial background of their patients is more remote.

The program of the meeting, at Del Monte, May 4, is as follows:

9:00 A.M.—President's Address—DR. R. T. LEGGE, Professor, Industrial Hygiene, University of California, Berkeley, California.

—Announcements by the Secretary—DR. CHRISTOFER LEGGO, Plant Physician, California and Hawaiian Sugar Refining Corporation, Ltd., Crockett, California.

—Demonstration of the Photo-Fluorographic Unit (35 MM)"—MR. HUGO CARLSON, District Mgr. Westinghouse Elec. and Mfg. Co., San Francisco, California.

—"Photo-Roentgenography in Industry"—DR. M. L. PINDELL, Roentgenologist, Los Angeles County Health Dept.

—"Industrial Physicians and the Defense Program"—DR. C. A. DUKES, Member of Committee on Medical Preparedness, American Medical Association.

—"The California Industrial Hygiene Program"—DR. J. P. RUSSELL, Chief, Industrial Hygiene Service, State of California, Berkeley.

Discussion.

12:30 P.M.—LUNCHEON.

2:00 P.M.—"The Accident, the Injury and the Surgeon's Report"—MR. CARL F. FRY, Chief, Bureau of Industrial Accident Prevention, State Industrial Accident Commission.

—"The Los Angeles Industrial Hygiene Program"—DR. V. A. NASATIR, Director, Division of Industrial Hygiene, Los Angeles City Health Dept.

—"Problems of Interest to the Industrial Physician"—DR. D. O. MCGOWAN, Plant Phys., General Motors Corporation, Los Angeles, California.

—"Diagnosis, Prevention, and Treatment of Occupational Dermatitis"—DR. LOUIS SCHWARTZ, Medical Director, Dermatoses Investigations, National Institute of Health, Bethesda, Maryland.

—"Toxicology of Newer Compounds in Industry"—DR. CHAUNCEY LEAKE, Ph.D., Prof. of Pharmacology, University of California, San Francisco, California.

Discussion.

Chicago Society

—1941 Meeting—

THE CHICAGO SOCIETY OF INDUSTRIAL MEDICINE AND SURGERY meets on May 20, 1941, in conjunction with the Annual Meeting of the Illinois State Medical Society, at the Palmer House, Chicago. DR. ROLLAND A. JACOBSON, President of the Society, announces the following program, prepared under the direction of DR. CHARLES DRUECK, JR., Program Chairman:

MORNING SESSION:

9:00—"Skin Lesions of the Hand" — THEODORE CORNBLAST, M.D., Ph.D., Ass't Professor, Department of Dermatology, University of Illinois.
Discussion.

9:45—"Fractures of the Pelvis" — GEORGE L. APFELBACH, M.D., F.A.C.S., Associate Professor of Surgery, Northwestern University.
Discussion.

10:30—"X-Ray Surveys of the Tuberculous Chest in Industry"—HOLLIS E. PORTER, M.D., Chicago.
Discussion.

11:15—"Disqualifying Conditions in Pre-employment Examination"—JAMES A. VALENTINE, M.D., and JOSEPH H. THOMAS, M.D.
Discussion.

AFTERNOON SESSION: (In conjunction with the Surgical Section of the Illinois State Medical Society):

"Fractures about the Elbow"—DR. JAMES J. CALLAHAN.

"The Use of Injections for the Relief of Peripheral Pain and Other Conditions"—DR. FREDERICK W. SLOBE.

Central States Society

—1941 Annual Meeting—

THE CENTRAL STATES SOCIETY OF INDUSTRIAL MEDICINE AND SURGERY will hold its 1941 Annual Meeting on May 13, at Davenport, Iowa, in conjunction with the meeting of the Iowa State Medical Society. The program of the General Session, Tuesday afternoon, May 13, and the evening session of the same day, is as follows:

SYMPOSIUM ON TRAUMA:

1:30-1:50—"Primary Care of Traumatic Wounds"—

Lewis M. Overton, M.D., Des Moines, Iowa.
Discussion.

2:00-2:20—"Traumatic Injuries of the Chest"—Willard Van Hazel, M.D., Associate Professor of Surgery, University of Illinois College of Medicine, Chicago.
Discussion.

2:30-2:50—"Peripheral Neuritis Following the Use of Serum"—John L. Garvey, M.D., Clinical Professor of Neurology, Marquette University, Milwaukee, Wisconsin.
Discussion.

3:00-3:20—"Fractures of the Patella Based on a Study of 554 Cases"—J. E. M. Thomson, M.D., Lincoln, Nebraska.
Discussion.

3:30-3:50—"Compensation Evaluation"—Oliver J. Fay, M.D., Des Moines, Iowa.
Discussion.

4:00-4:20—"Early Management of Fractures of the Long Bones"—William R. Cubbins, M.D., Clinical Professor of Bone and Joint Surgery, Loyola University School of Medicine, Chicago.
Discussion.

Evening Session:

6:30—"The Relationship of the New Antiseptics to the Treatment of Infected Wounds and Septicemia"—H. Winnett Orr, M.D., Lincoln, Nebraska.
Discussion.

Both of the sessions will be held in the Main Ball Room of Hotel Blackhawk. Dr. Roland A. Jacobson, of Chicago, and Dr. Donald C. Connsett, of Dubuque, Iowa, will be co-chairmen.

Tuberculosis in Industry

—Saranac Lake Symposium—

THE Symposium on Tuberculosis in Industry, to be held at Saranac Laboratory for the Study of Tuberculosis, Saranac Lake, New York, June 9 to 14, 1941, under the sponsorship of the Trudeau School of Tuberculosis, has the following program:

Monday, June 9:

MORNING: The Disease, Tuberculosis.
Registration: Mr. Roy Dayton, Secretary.
Introduction: Dr. Leroy U. Gardner, Saranac Laboratory.
Tuberculous Infection, Sources and Modes of Transmission—Dr. Leroy U. Gardner.
Pathology—Dr. Arthur J. Vorwald, Saranac Laboratory.
Diagnosis—Dr. Francis B. Trudeau, Saranac Laboratory.
Treatment and Rehabilitation—Dr. Fred H. Heise, Trudeau Sanatorium.
Epidemicology—Dr. L. L. Lumsden, U. S. Public Health Service.

AFTERNOON: Incidence of Tuberculosis in Industrial Groups.
Comparative Mortality Statistics on Tuberculosis in Various Industries—Dr. Louis I. Dublin, Metropolitan Life Insurance Company.
Disability Morbidity from Tuberculosis—Dr. W. M. Gafafer, U. S. Public Health Service.

Tuesday, June 10:

MORNING: Predisposing Factors, Theoretically Significant.
1. Non-Specific Factors:
(a) Heredity, Race, Age, Sex, Selective Employment, Economic Status—Miss Jessamine Whitney, National Tuberculosis Association.
(b) Influence of Foreign Birth—Dr. O. A. Sander, Milwaukee, Wisconsin.
(c) Influence of Nutrition—Dr. J. A. Johnston, Henry Ford Hospital.

AFTERNOON: 2. "Specific" Occupational Factors:
(a) Respiratory Irritants: Dusts, Fumes, and Gases—Dr. Leroy U. Gardner.
(b) Lead Absorption and Lead Intoxication — Dr. Elston L. Belknap, Milwaukee, Wisconsin.

Wednesday, June 11:

MORNING: 2. "Specific" Occupational Factors (Cont.):
(c) Influence of Fatigue as Observed in the Automobile Industry—Dr. Fred B. Wishard, General Motors Corporation.
(d) Influence of Temperature and Humidity — Dr. Anna M. Baetjer, Johns Hopkins School of Hygiene and Public Health.
(e) Influence of Posture and Trauma — Dr. E. N. Packard, Saranac Lake, New York.

AFTERNOON: Incidence and Causation of Tuberculosis, Various Industries.
1. Office Workers — Dr. Haynes Harold Fellows, Metropolitan Life Insurance Company.
2. Tobacco and Cigar Makers — Dr. Charles-Francis Long, Bayuk Cigars Incorporated.

Thursday, June 12:

MORNING: Various Industries (Cont.).
3. Garment and Laundry Workers — Dr. Herbert R. Edwards, New York City Department of Health.
4. Cement and Gypsum Workers—Dr. D. M. Brumfiel, Saranac Laboratory.
5. Grain Handlers—Dr. Adelaide Ross Smith, New York State Department of Labor.

AFTERNOON: 6. Iron and Steel Workers—Dr. A. G. Kammer, Inland Steel Company.
7. Foundrymen—Dr. Leonard Greenburg, New York State Department of Labor. Dr. Alton S. Pope, Massachusetts Department of Public Health.

Friday, June 13:

MORNING: Various Industries (Cont.).
8. Granite vs. Marble Cutters—Dr. Louis Benson, Vermont Sanatorium.
9. Hard Rock Miners—Mr. Donald E. Cummings, Saranac Laboratory.
10. Medical Students, Nurses, and Laboratory Workers—Dr. J. Burns Amherson, Bellevue Hospital.

AFTERNOON: Methods of Control.
Administrative Procedures Within the Plant—Dr. William A. Sawyer, Eastman Kodak Company.
Public Health Relationships—Dr. Leverett D. Bristol, American Telephone and Telegraph Company.
Case Finding Procedures — Dr. William Siegal, New York State Department of Health.

Saturday, June 14:

MORNING: The Viewpoint of the Insurance Carrier on Tuberculosis in Industry — Mr. Henry D. Sawyer, Compensation Insurance Rating Board.
The Viewpoint of a State Compensation Commissioner—Mrs. Emma S. Toussant, Massachusetts Department of Industrial Accidents.
A Labor Viewpoint of Tuberculosis in Industry — Mr. Matthew Woll, American Federation of Labor.
Closing Remarks—Dr. Leroy U. Gardner.

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Industrial Medicine
Reg. U. S. Pat. Off.

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Volume 10 **MAY, 1941** **Number 5**

"In All Good Will"

NO pleasanter or more profitable occasion could be recommended to any physician or surgeon than that afforded by the Pittsburgh meetings of the AMERICAN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS and the AMERICAN INDUSTRIAL HYGIENE ASSOCIATION May 5 to 9, 1941. The program of the meetings (in full on pages 205-6-7) is imposing and important in its mere recital of subjects and names. It is the one because of the scope and diversity of the matters to be presented and the intensity of study and depth of experience crystallized in each presentation. The other because of the scope and diversity of the present-day industrial problems not only of the industrial physician but also of the general practitioner. The sessions well deserve their characterization as a post-graduate "Institute of Industrial Medicine and Industrial Hygiene." They condense into five days a wealth of material that will not have outlived its usefulness in several times that many years, and will be of particular value to the numerous medical men whose contacts with industrial medicine, more or less casual in the past, are certain to become vastly more intimate in the immediate future.

The AMERICAN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS arrives at its meeting with a steadily growing record, a rapidly growing membership, and a tremendously growing opportunity and responsibility. This is its twenty-sixth annual convention, and its membership is now close to 1300. Its opportunity, however, is not so quickly summarized.

Industrial medicine, in its creditable past having pretty well taken the measure of the curative and reparative, and being well along in the preventive, so far as the individual employee is concerned, now finds itself also on the threshold of the working environment, about to become indispensable in the establishment and maintenance of the conditions of industrial health for all employees. Those who have been in industrial medicine a long time know how much that involves. It is a large undertaking under any circumstances. Under the pressure of the immediate requirements it is almost colossal. Moreover, in any estimate of the shape of things to come, it grows and grows. The future of industrial medicine, and the opportunity of the members of the Association—who *are* industrial medicine—lie within it.

The same is true of the coordinate organization, the AMERICAN INDUSTRIAL HYGIENE ASSOCIATION. It comes to its second annual meeting with a membership considerably enlarged during the year, and with an equal opportunity and responsibility in the new and greater problem of environment. It brings the technical skill to meet any situation which that problem may present. Comparatively young as an organization, it makes up in vigor and ability what it may not have in years. Its members *are* industrial hygiene.

Omen of the successful realization of their opportunity and responsibility on the part of both Associations is the remarkable record of what their united efforts have already brought about within the short period of the recent pressure. Advances have been numerous and rapid, and, though the pace is still accelerating, the industrial physician and the industrial hygienist are still in the front rank, and neither is out of breath.

Their combined program, as above mentioned, is eloquent of the diversity of their problems and convincing of the fact of solution—not only as its 56 presentations will be given to be heard, but also as they will be published to be read, and studied, in this the official Journal of both Associations.

The fact of subsequent publication, however, should not prevent attendance. Every paper on the program should be heard *and* read. Not only because of the intrinsic values which more and more appear, but also because in the reading it is pleasant to recall the circumstances, the personal equation of the hearing.

There is something warm and compelling about the meetings of these Associations. Whatever it is, it must be deeply rooted in the very nature of industrial practice. So deeply rooted, indeed, that it appears in the first printed thesis dealing with occupational health. It is expressed in the foreword of a treatise by Ulrich Ellenbog (p. 204).

"Therefore I do not wish to withdraw myself and remain aloof to the masters of this trade, but to serve them by giving assistance and advice in order that they may not receive too severe an illness which is produced by these fumes. I beg of you with all my heart to take this, my modest advice, written with my own hand in all good will and friendship. Nothing more would I desire than to serve each and all with friendly service."

That is the spirit of industrial medicine!

INDUSTRIAL HEALTH AND DISEASE

Industrial Health and National Defense

LEVERETT D. BRISTOL, M.D., DR.P.H.,
New York

AN ADEQUATE national defense requires a vigorous health offensive! Stimulated by the launching of the nation's defense program, the organized medical and public health professions have been quick to realize the importance of the contributions which they can make to health preparedness. The mobilization of the medical profession for any emergency that may arise has been successfully inaugurated with the appointment of a National Committee on Medical Preparedness by the American Medical Association.

Man power is now the greatest need both of industrial production and of military defense. Adequate man power is dependent on the highest type of mental and physical fitness; nor should the health of women workers be forgotten in these times, in view of their important contributions to industrial production and operation and their possible substitution for men in times of serious national emergency. The speed-up of production and manufacture, the development of new material and methods, the increased employment of new workers unused to the hazards of industry will require new industrial health routines and regulations, more intensive study and research in the field of occupational diseases and industrial poisons and the extension and improvement of all health and safety education. To speed the work but to spare the worker should be our aim!

Read before the Section on Preventive and Industrial Medicine and Public Health at the Ninety-First Annual Session of the American Medical Association, New York, June 13, 1940; published in *J.A.M.A.*, October 12, 1940.

Read before the International Society of Medical Health Officers, Detroit, October 7, 1940; published in *J.A.M.A.*, October 12, 1940.

General Health Preparedness

INDUSTRIAL health preparedness in the United States is only one phase of a general health preparedness program, although from the standpoint of national defense industrial health undoubtedly should command first attention, since it involves our adult civilian population of productive workers and the enlisted land, sea and air forces of the country, on all of which successful national defense in times of emergency must depend. The first essential in our countrywide public health program during these times of preparation for adequate national defense is a thorough scanning of our organized medical and health machinery to be sure that important matters of coordination and administration are provided and that there are no gaps or weaknesses in Federal, state, county or city health administration or in public and private medical facilities. Moreover, during times of temporary national emergency, centralization of medical and public health authority is required.

Industrial Health

DURING the World War of 25 years ago I had the privilege of serving as commissioner of health of one of the New England states. Our work was closely integrated with that of the Federal Public Health Service during the war period. In fact, the Surgeon General of the Public Health Service suggested that all state health officers remain at their posts in order to cooperate in the federal wartime health program. The one thing I do not remember as an important part of our Federal-state program at that time was industrial health or industrial medicine. The fact is that private industrial medicine in this country

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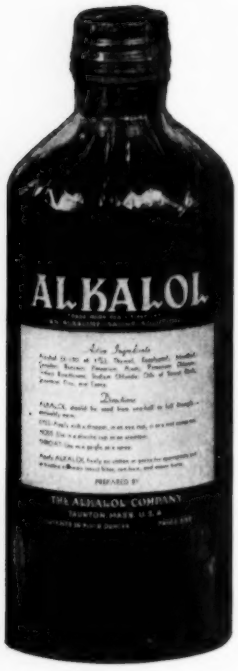

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was then only in "swaddling clothes" and one or two industrial hygiene units in state governments had only just been born. The Division of Industrial Hygiene of the United States Public Health Service and the Section on Industrial Hygiene of the American Public Health Association are only a little over 25 years old, having been created in 1914. The American Association of Industrial Physicians and Surgeons also is but 25 years old, while the American Industrial Hygiene Association held its first annual meeting this past June. It has been only during the past three or four years that bureaus of industrial hygiene have been rapidly extended in health departments of various states and cities, under the excellent leadership of the United States Public Health Service. Only since 1937 has the Council on Industrial Health of the American Medical Association been operative in cooperation with committees or activities on industrial health in 34 state medical societies, most of which have been organized during the past three years. In other words, the specialty of industrial health as we know it in this country is at present only a little over 25 years old and has developed almost entirely since the World War.

As a part of a national defense program, it becomes evident from the foregoing facts that industrial physicians and industrial hygienists of this country have little in the way of precedent or wartime experience on which to base their efforts, unless it be, to some extent, the recruiting experience and a few engineering experiences during the World War. As a matter of record, it may safely be predicted that the present national preparedness program will be to industrial medicine and industrial hygiene in the United States what the World War was to the greater stimulation of interest and plans in the field of social hygiene. It is particularly to be hoped that the present plans for national defense will result in more active cooperation between industrial medical departments and local and state health departments and medical societies. One concrete achievement of such coordinated efforts might be a much needed national plan for standardized recording and reporting of industrial morbidity and mortality, particularly of occupational diseases.

Private Industrial Medicine

ONE of the most important problems common to industrial medicine and military medicine is that of traumatic surgery. Industrial medicine's so-called parents might be said to have been (1) traumatic injuries of workers and (2) workmen's compensation laws. In any national preparedness program much should be done to increase the facilities for teaching traumatic surgery in our medical schools and in developing post-graduate instruction for practicing physicians through county and state medical societies and military organizations. In this connection, much of the leadership in promoting and teaching traumatic surgery might be available from those industrial surgeons who even during peace time have become experts in the fields of traumatic surgery and first aid in accidents and infections.

Industrial medicine, even in a national defense program, must be preventive and educational as well as reparative, reconstructive and curative. It must concern itself with nonoccupational as well as occupational diseases. Industrial physicians will have important parts to play in the national defense program from the standpoints both of (a) the physical and mental fitness of civilian workers and enlisted forces and of (b) the safety and healthfulness of working and training environments.

During the present national emergency, professional facilities must be made available not only for a successful medicomilitary program but also for the continuity of adequate medical care for the civilian working population. In the latter connection it should be stressed that much of the load of responsibility will continue to be carried by individual practicing physicians. The great

bulk of industrial medical work is now being carried on by private physicians on a part time basis. One of the first essentials, therefore, in an industrial health preparedness program is to determine all physicians in the United States who are qualified for industrial medical service, with some indication of their experience and special fields, such as administration, examinations, first aid, industrial surgery, toxicology and industrial health education.

The possible availability of properly qualified full time industrial physicians and surgeons for special assignments in the federal service should be given careful attention while at the same time acknowledging the fact that as a group the greatest usefulness of trained industrial physicians in a national emergency may be in their remaining at their posts in private industrial organizations. The inauguration of a plan for state and regional consultants on industrial health would be desirable in the interest of better organization and coordination. Such consultants, during the preparedness emergency, might be attached to the United States Public Health Service. Their chief function would be as consultants and coordinators working in cooperation with (a) state and local health officers, (b) state and county medical society committees on industrial health, and (c) industrial medical departments.

The Council on Industrial Health of the American Medical Association at its last meeting expressed its willingness to assist in the development of intensive training on industrial health methods for full time and part time industrial physicians, through its organization of committees in state medical societies. The Council already has acquired information on all major medical agencies interested in employee health and on the available facilities for undergraduate and postgraduate industrial medical training in the educational institutions of the United States, as well as other data of potential value in an industrial health preparedness program as a part of national defense. A general statement of medical relationships in industry and of industrial medical education appeared in a special industrial health issue of *J.A.M.A.* last February.[1] Speaking as a member of the Council, which is a standing committee of the Board of Trustees of the American Medical Association, we would welcome the cooperation of the International Society of Medical Health Officers in all matters pertaining to the advance of industrial health.

Objectives

THE basic objectives of both industrial medicine and military medicine are much the same, namely:

1. To ascertain, by examination, the fitness of individuals for civilian work or military service, and to place individuals in activities consonant with their physical and mental assets and liabilities.

2. To maintain and improve the health and efficiency of those already employed or in government service.

3. To educate the worker or the enlisted man on matters pertaining to personal hygiene.

4. To reduce lost time and absenteeism from industrial occupation or military duty because of illness or injury.

To reach these objectives of either industrial or military medicine a definite plan or program must be formulated under suitable medical direction, with the employment of qualified physicians, surgeons, nurses and other specialists and technicians as may be needed. Medical offices and adequate emergency, dispensary and hospital facilities are essential. Suitable physical examination and medical records are necessary. Medical supervision also should include all matters of general sanitation and environmental hygiene, with the assistance of specialists, such as engineers and chemists, as required.

Physical Examinations

OF PRIMARY importance among the objectives common to industrial and military medicine are physical examinations. The scope or ex-

1. Industrial Health, *J.A.M.A.*, 114:573, February 17, 1940.

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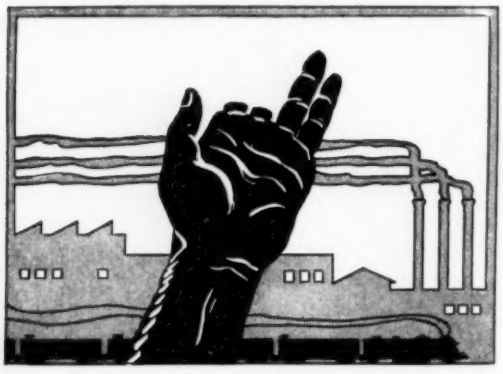

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tent of preplacement examinations must be based largely on the character and general conditions and needs of the industry or the military assignment. Such examinations may vary all the way from a very brief, superficial physical inspection to a detailed medical examination of an hour or more. We shall have to strike some midground as far as the average examination is concerned. We certainly want to make examinations that are worth something; on the other hand, there is a limit that may be reached as to their practicability from the standpoints of time and expense. Physical tests of employees during the national defense emergency might possibly be based on requirements and record forms previously agreed on jointly by military and industrial authorities.

The purpose of medical examinations should not be to keep people out of work or out of military service but rather to know what the actual conditions are and to make certain that the individual does not take up a job for which he is not fitted. Time will not permit of a detailed account of the various types of physical examination procedures and forms of examination classifications or of acceptance and placement standards and causes for rejection. These subjects have been extensively covered from time to time in pamphlets and bulletins.

As interest grows in the subject of mental hygiene, which underlies the whole realm of physical health and safety, more and more attention must be given to mental and nervous system examinations in connection with routine physical appraisals of civil and military groups. Mad mental hygiene of individuals and masses to a large degree is responsible for much industrial unrest and even for lowered morale in armed forces. To obviate this the promotion of a sound program of mental health in industry and among enlisted forces is the only alternative; this, in turn, can be built only on knowledge obtained through satisfactory mental inventories of employee or recruit groups.

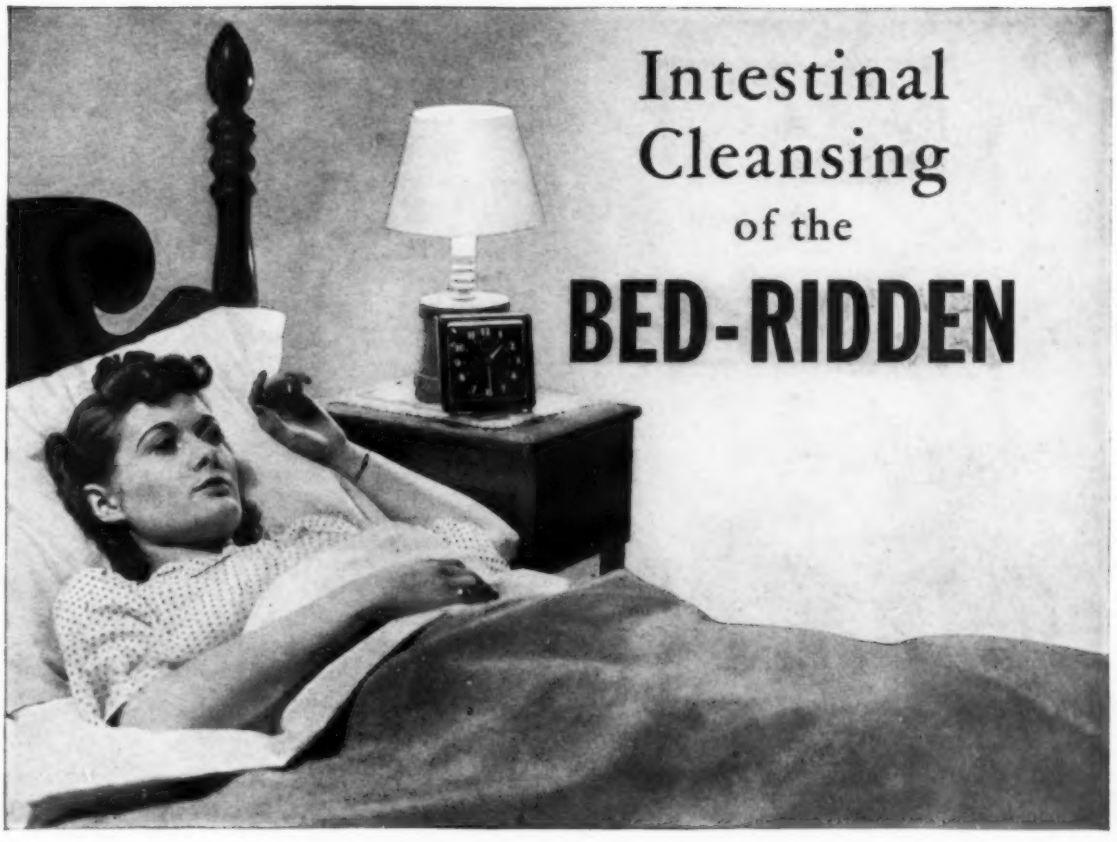
Of all measures of industrial health preparedness from the standpoint of national defense there is none more important than the periodic health examination of individuals in working groups. Such examinations should serve not only (a) to guard the health of civilian employees in industry, particularly those in the so-called war industries, from excessive fatigue and the possible ill results of speed, pressure and special hazards associated with national preparedness but also (b) to maintain a high level of physical and mental fitness in those private industrial "reservoirs" which at any time might have to be drawn on for government service. Business organizations may thus contribute to national defense by encouraging such health examinations among their employees. Physical examinations, both initial and periodic, fall short of their full value unless followed up with programs for the control and correction of incipient diseases and impairments which are found. Moreover, these examinations, above all, should be considered as important educative experiences.

Public Industrial Hygiene

PRIOR to 1936 there were in the United States only five state departments of health and two or three state departments of labor conducting industrial hygiene activities, and even these activities were of limited nature. As of 1940, a total of 39 state and local industrial hygiene units have been established in 29 states and two territories, mostly in departments of health. Some of these are of very recent creation and have only skeleton organizations. There is great need that these be strengthened as rapidly as possible as a part of the national industrial health preparedness program. Moreover, additional industrial hygiene units should be organized without delay in the remaining states and territories and additional local governments, particularly of the larger cities. Steps should be taken to make possible the financing of these industrial hygiene units by state and local appropriations. At present nearly 80% of funds budgeted for public industrial hygiene is contributed by the federal government. During the present emergency, additional funds should be provided to the Public Health Service to train additional industrial hygiene personnel. Care should be taken to emphasize that such industrial hygiene activities are properly the function of health departments and to discourage steps taken in a few localities to develop such essential health services in other agencies.

Conclusion

IT MAY be stated that industrial physicians and industrial hygienists, like medical public health officers, are ready and willing to serve their country in this preparedness emergency. Industrial health work should be a strong arm in national defense, particularly in the preservation of expert workers and assisting in the development of additional skilled workers. Reducing lost time due to occupational and nonoccupational illness and accidents and cutting down excessive exposure to injurious materials are part of the industrial health program which will be conducive to maximum national effectiveness and production. It is particularly necessary that studies be made to determine unfavorable environmental factors in the production of war equipment and munitions. The business employer or the governmental unit that considers reducing its industrial health program at this time, because of what at the moment may seem to be more important activities, is most unwise. As a matter of fact, the needs of national defense require a strengthening and broadening of the entire industrial health program all along the line from private industry to local, state and federal jurisdictions. In this significant period of rearmament, during which our chief defense will be our industrial skill, let us be sure that we place first on our list the health and safety conservation of our industrial workers.



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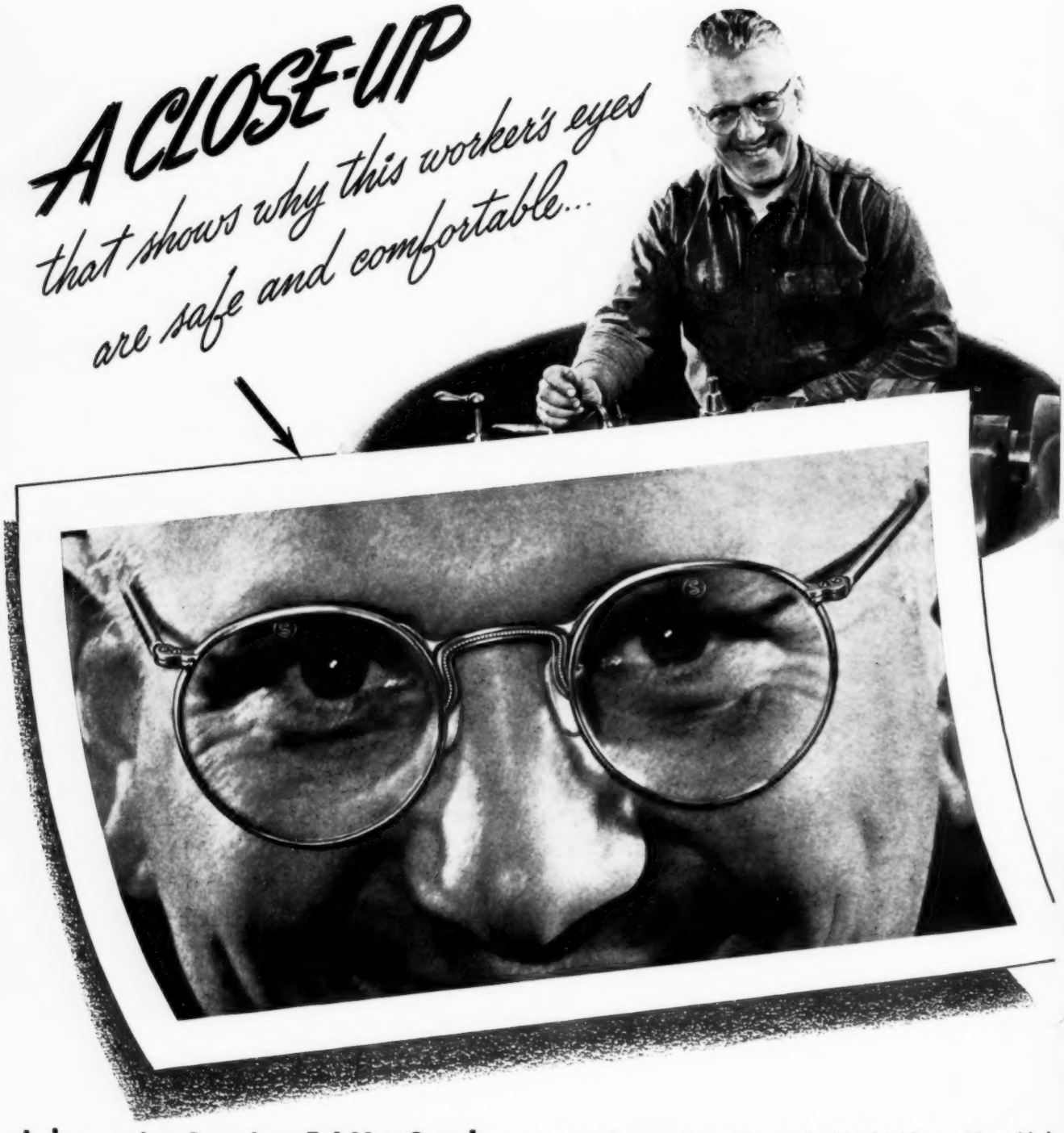

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